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# MEDICO-CHIRURGICAL TRANSACTIONS

PUBLISHED BY

THE ROYAL  
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OF  
LONDON

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LONDON

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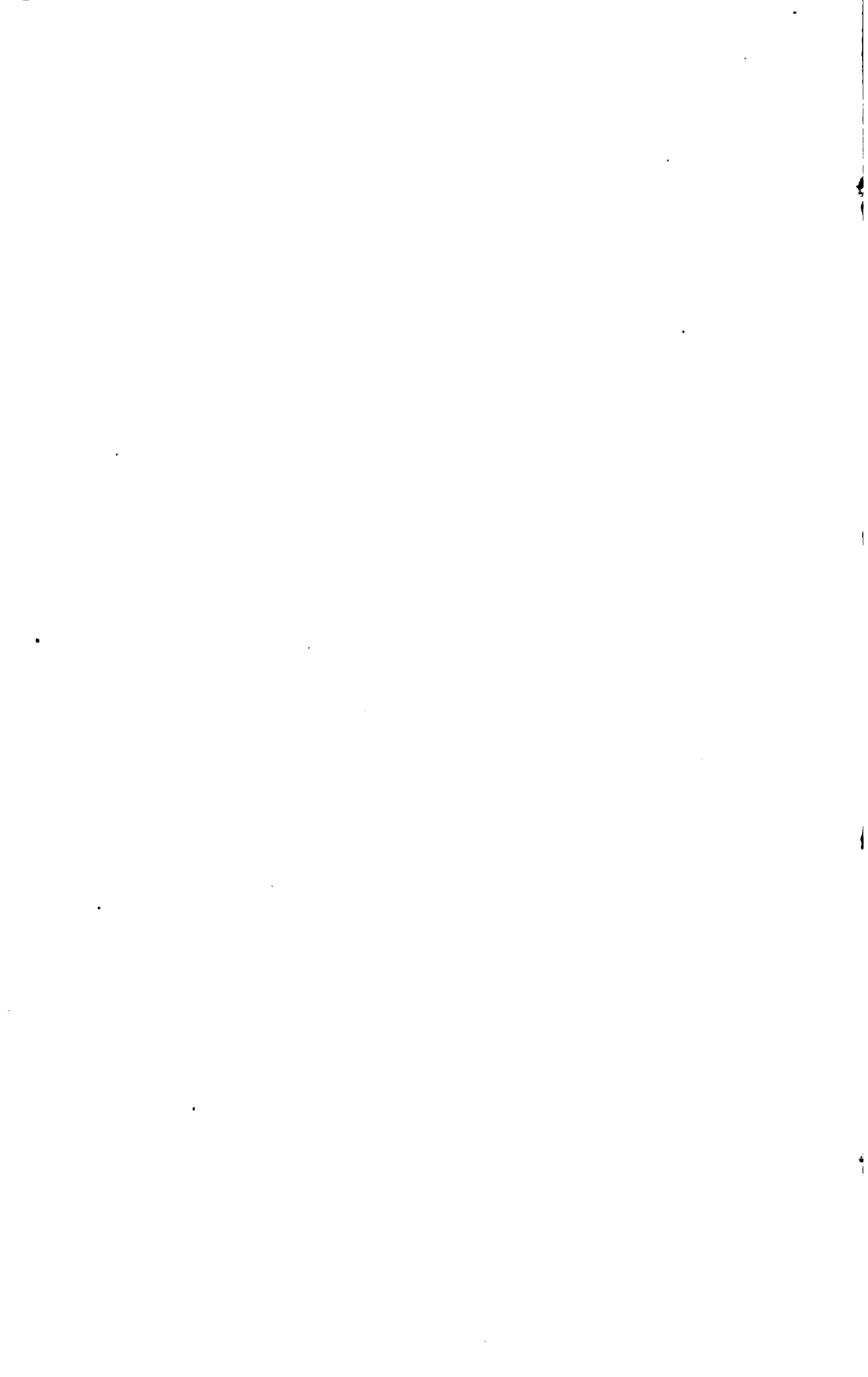
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*August, 1895.*

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# ROYAL MEDICAL AND CHIRURGICAL SOCIETY OF LONDON

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PATRON  
**THE QUEEN**

---

OFFICERS AND COUNCIL  
ELECTED MARCH 1, 1895.

---

*President*

JONATHAN HUTCHINSON, F.R.S.

VICE-PRESIDENTS	{	CHARLES JOHN HARE, M.D. JOHN HARLEY, M.D. *JOHN WARRINGTON HAWARD. JOHN LANGTON.
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HON. SECRETARIES	{	JOHN MITCHELL BRUCE, M.D. ROBERT WILLIAM PARKER.
HON. LIBRARIANS	{	SAMUEL JONES GEE, M.D. RICKMAN J. GODLEE, M.S.
OTHER MEMBERS OF COUNCIL	{	WILLIAM EWART, M.D. FREDERICK THOMAS ROBERTS, M.D. FELIX SEMON, M.D. FREDERICK TAYLOR, M.D. FRANCIS CHARLEWOOD TURNER, M.D. ARTHUR EDWARD J. BARKER REGINALD HARRISON JOHN HAMMOND MORGAN FREDERICK TREVES THOMAS FITZ-PATRICK, M.D.

\* Mr. Bostock died on May 18th, and Mr. J. Warrington Haward was elected to succeed him on June 11th, 1895, vacating at the time the office of Vice-President.

# FELLOWS OF THE SOCIETY APPOINTED BY THE COUNCIL AS REFEREES OF PAPERS

FOR THE SESSION OF 1895-6.

---

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---

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[The President and Hon. Secretaries are *ex officio* members of all Committees.]

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G. V. POORE, M.D.

A. Q. SILCOCK, B.S.

J. KNOWSLEY THORNTON, C.M.

### COMMITTEE

*Appointed to investigate the Medical Climatology and Balneology of Great Britain and Ireland.*

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HERMANN WEBER, M.D.

C. THEODORE WILLIAMS, M.D.

ARCHIBALD E. GARROD, M.D., *Hon. Sec.*

### COMMITTEE

*Appointed to investigate the subject of Suspended Animation in the Drowned.*

EDWARD ALBERT SCHÄFER, F.R.S., *Chairman*

GEORGE HARLEY, M.D., F.R.S.

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THOMAS PICKERING PICK, *Hon. Sec.*

### HOUSE COMMITTEE

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A. PEARCE GOULD, M.S.

J. WARRINGTON HAWARD

TIMOTHY HOLMES

ALFRED WILLETT

# PRESIDENTS OF THE SOCIETY FROM ITS FORMATION AS THE MEDICO-CHIRURGICAL SOCIETY, 1805

## ELECTED

1805. WILLIAM SAUNDERS, M.D.
1808. MATTHEW BAILLIE, M.D.
1810. SIR HENRY HALFORD, BART., M.D., G.C.H.
1813. SIR GILBERT BLANE, BART., M.D.
1815. HENRY CLINE
1817. WILLIAM BABINGTON, M.D.
1819. SIR ASTLEY PASTON COOPER, BART., K.C.H., D.C.L.
1821. JOHN COOKE, M.D.
1823. JOHN ABERNETHY
1825. GEORGE BIRKBECK, M.D.
1827. BENJAMIN TRAVERS
1829. PETER MARK ROGET, M.D.
1831. SIR WILLIAM LAWRENCE, BART.
1833. JOHN ELLIOTSON, M.D. (First President of the Society  
after its Incorporation as the Royal Medical and  
Chirurgical Society of London, 1834).
1835. HENRY EARLE
1837. RICHARD BRIGHT, M.D., D.C.L.
1839. SIR BENJAMIN COLLINS BRODIE, BART., D.C.L.
1841. ROBERT WILLIAMS, M.D.
1843. EDWARD STANLEY
1845. WILLIAM FREDERICK CHAMBERS, M.D., K.C.H.
1847. JAMES MONCRIEFF ARNOTT
1849. THOMAS ADDISON, M.D.
1851. JOSEPH HODGSON
1853. JAMES COPLAND, M.D.
1855. CÆSAR HENRY HAWKINS
1857. SIR CHARLES LOCOCK, BART., M.D.
1859. FREDERIC CARPENTER SKEY
1861. BENJAMIN GUY BABINGTON, M.D.
1863. RICHARD PARTRIDGE
1865. SIR JAMES ALDERSON, M.D., D.C.L.
1867. SAMUEL SOLLY
1869. SIR GEORGE BURROWS, BART., M.D., D.C.L.
1871. THOMAS BLIZARD CURLING
1873. CHARLES JAMES BLASIUS WILLIAMS, M.D.
1875. SIR JAMES PAGET, BART., D.C.L., LL.D.
1877. CHARLES WEST, M.D.
1879. JOHN ERIC ERICHSEN
1881. ANDREW WHYTE BARCLAY, M.D.
1882. JOHN MARSHALL
1884. SIR GEORGE JOHNSON, M.D.
1886. GEORGE DAVID POLLOCK
1888. SIR EDWARD HENRY SIEVEKING, M.D., LL.D.
1890. TIMOTHY HOLMES
1892. SIR ANDREW CLARK, BART., M.D., LL.D., F.R.S.  
(*Sir Andrew Clark died 6th November, 1893, and Dr. W. S.  
Church, Senior Vice-President, officiated as President  
until the following 1st March, 1894.*)
1894. JONATHAN HUTCHINSON, F.R.S.

## HONORARY FELLOWS

(Limited to Twelve.)

### *Elected*

- 1887 FLOWER, SIR WILLIAM HENRY, K.C.B., LL.D., F.R.S.,  
Director of the Natural History Department, British  
Museum, Cromwell road.
- 1887 FOSTER, MICHAEL, M.D., LL.D., F.R.S., Professor of  
Physiology in the University of Cambridge.
- 1883 FRANKLAND, EDWARD, M.D., D.C.L., Ph.D., F.R.S., Cor-  
responding Member of the Academy of Sciences of  
France; The Yews, Reigate Hill, Reigate.
- 1868 HOOKER, SIR JOSEPH DALTON, C.B., M.D., K.C.S.I., D.C.L.,  
LL.D., F.R.S., Corresponding Member of the Academy  
of Sciences of France; The Camp, Sunningdale.
- 1878 LUBBOCK, the Right Hon. SIR JOHN, Bart., M.P., D.C.L.,  
LL.D., F.R.S., High Elms, Farnborough, Kent, R.S.O.
- 1873 STOKES, SIR GEORGE GABRIEL, Bart., M.A., D.C.L., LL.D.,  
F.R.S., M.P., Lucasian Professor of Mathematics in  
the University of Cambridge; Lensfield Cottage,  
Cambridge.
- 1867 TURNER, SIR WILLIAM, M.B., D.C.L., LL.D., F.R.S., Pro-  
fessor of Anatomy in the University of Edinburgh;  
6, Eton Terrace, Edinburgh.

## FOREIGN HONORARY FELLOWS

(Limited to Twenty.)

### *Elected*

- 1878 BACCELLI, GUIDO, M.D., Professor of Medicine at Rome.
- 1887 BILLINGS, JOHN S., M.D., D.C.L. Oxon., Surgeon U.S. Army ;  
Librarian, Surgeon-General's Office, Washington.
- 1883 DuBois REYMOND, EMIL, M.D., Professor of Physiology,  
Berlin ; Neue Wilhelmstrasse 15, Berlin.
- 1887 VON ESMARCH, FRIEDRICH, M.D., Professor of Surgery in  
the University of Kiel.
- 1868 KÖLLIKER, ALBERT, Professor of Anatomy in the University  
of Würzburg.
- 1868 LARREY, HIPPOLYTE BARON, Member of the Institute of  
France ; Inspector of the "Service de Santé Militaire,"  
and Member of the "Conseil de Santé des Armées ;"  
Commander of the Legion of Honour, &c. ; Rue de  
Lille, 91, Paris.
- 1883 PASTEUR, LOUIS, LL.D., Member of the Institute of France.
- 1856 VIRCHOW, RUDOLPH, M.D., LL.D., Professor of Patho-  
logical Anatomy in the University of Berlin ; Corre-  
sponding Member of the Academy of Sciences of  
France ; 10, Schellingstrasse, Berlin.

# FELLOWS

OF THE

## ROYAL MEDICAL AND CHIRURGICAL SOCIETY

### OF LONDON

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#### EXPLANATION OF THE ABBREVIATIONS

P.—President.	C.—Member of Council.
V.P.—Vice-President.	<i>Sci. Com.</i> —A Scientific Committee.
T.—Treasurer.	<i>Ho. Com.</i> —House Committee.
L.—Hon. Librarian.	<i>Lib. Com.</i> —Library Committee.
S.—Hon. Secretary.	<i>Bldg. Com.</i> —Building Committee.

The abbreviations *Trans.* and *Pro.*, followed by figures, show the number of Papers which have been contributed to the 'Transactions' or 'Proceedings' by the Fellow whose name they follow. *Referee*, *Sci. Com.*, and *Lib. Com.*, *Bldg. Com.*, *Ho. Com.*, with the dates of office, are attached to the names of those who have served as Referees of papers and on the Committees of the Society.

Those marked thus (†) have paid the Composition Fee in lieu of further annual subscriptions.

Those marked thus (\*) have paid the Composition Fee entitling them to receive the 'Transactions.'

---

#### RESIDENT FELLOWS

##### *Elected*

†1877 ABERCROMBIE, JOHN, M.D., Physician to, and Lecturer on Forensic Medicine at, Charing Cross Hospital; 23, Upper Wimpole street, Cavendish square. *Trans.* 1.

1885 ABRAHAM, PHINEAS S., M.A., M.D., Lecturer on Physiology and Histology at the Westminster Hospital; 2, Henrietta street, Cavendish square.

*Elected*

- 1885 ACLAND, THEODORE DYKE, M.D., Physician to St. Thomas's Hospital, and Physician to the Hospital for Consumption and Diseases of the Chest, Brompton ; 74, Brook street, Grosvenor square, W.
- †1852 ADAMS, WILLIAM, Consulting Surgeon to the Great Northern Central Hospital, the National Hospital for the Paralysed and Epileptic, and the National Orthopædic Hospital ; 5, Henrietta street, Cavendish square. C. 1873-4. *Trans.* 3.
- 1867 AIKIN, CHARLES ARTHUR, 12, Ladbroke terrace, Notting hill.
- 1879 ALLCHIN, WILLIAM HENRY, M.D., F.R.S. Ed., Physician to the Westminster Hospital ; 5, Chandos street, Cavendish square.
- 1890 ALLINGHAM, HERBERT WILLIAM, Assistant Surgeon to St. George's Hospital ; 25, Grosvenor street, Grosvenor square.
- †1863 ALTHAUS, JULIUS, M.D., Consulting Physician to the Hospital for Epilepsy and Paralysis, Regent's Park ; 48, Harley street, Cavendish square. *Trans.* 2.
- 1888 ANDERSON, JOHN, M.D., C.I.E., Physician to the Seamen's Hospital, Greenwich ; 9, Harley street, Cavendish square.
- 1890 ANDERSON, WILLIAM, Surgeon to St. Thomas's Hospital ; Professor of Anatomy to the Royal Academy of Arts ; 2, Harley street, Cavendish square.
- 1891 ANDREWES, FREDERICK WILLIAM, M.B., 35, Welbeck street, Cavendish square.
- 1891 ANDREWS, LAUNCELOT, M.B., 22, Cheyne gardens, Manor street, Chelsea.
- 1888 ARKLE, CHARLES, M.D., Assistant Physician to Charing Cross Hospital ; 66, Wimpole street, Cavendish square.
- 1893 BAILEY, ROBERT COZENS, M.S., 21, Welbeck street, Cavendish square.
- 1887 BALL, JAMES BARRY, M.D., Physician to the West London Hospital ; 12, Upper Wimpole street, Cavendish square.



*Elected*

- 1885 BALLANCE, CHARLES ALFRED, M.S., Assistant Surgeon to St. Thomas's Hospital and to the Hospital for Sick Children, Great Ormond street; Surgeon to the National Hospital for the Paralysed and Epileptic, Queen square; 106, Harley street, Cavendish square. *Trans.* 1.
- 1879 BARKER, ARTHUR EDWARD JAMES, Professor of the Principles and Practice of Surgery and Professor of Clinical Surgery at University College, and Surgeon to University College Hospital, London; 87, Harley street, Cavendish square. C. 1895. *Trans.* 7.
- †1876 BARLOW, THOMAS, M.D., B.S., *Trustee for Debenture-holders*; Physician to University College Hospital, and to the Hospital for Sick Children, Great Ormond street; 10, Wimpole street, Cavendish square. C. 1892. *Trans.* 2.
- 1893 BARRETT, HOWARD, 49, Gordon square.
- 1880 BARROW, A. BOYCE, Surgeon to King's College Hospital; 37, Wimpole street, Cavendish square.
- 1859 BARWELL, RICHARD, Consulting Surgeon to the Charing Cross Hospital; 55, Wimpole street. C. 1876-77. V.P. 1883-4. *Referee*, 1868-75, 1879-82. *Trans.* 12.
- †1868 BASTIAN, HENRY CHARLTON, M.A., M.D., F.R.S., Professor of Medicine in University College, London; Physician to University College Hospital and to the National Hospital for the Paralysed and Epileptic; 8A, Manchester square. C. 1885. *Referee*, 1886—. *Trans.* 2.
- 1890 BATEMAN, WILLIAM A. F., Bridge House, Richmond, Surrey.
- 1891 BATTEN, FREDERICK E., M.A., M.B., B.C.Cantab., 124, Harley street.
- 1875 BEACH, FLETCHER, M.B., Winchester House, Kingston Hill [64, Welbeck street, W.].
- 1883 BEALE, EDWIN CLIFFORD, M.A., M.B., Physician to the City of London Hospital for Diseases of the Chest, and Physician to the Great Northern Central Hospital; 23, Upper Berkeley street.

*Elected*

- 1862 BEALE, LIONEL SMITH, M.B., F.R.S., Professor of the Principles and Practice of Medicine in King's College, London, and Physician to King's College Hospital; 61, Grosvenor street. C. 1876-7. *Referee*, 1873-5. *Trans.* 1.
- 1880 BEEVOR, CHARLES EDWARD, M.D., Physician for Out-patients to the National Hospital for the Paralysed and Epileptic, and to the Great Northern Hospital; 33, Harley street, Cavendish square. *Trans.* 1.
- 1883 BENNETT, STORER, Dental Surgeon to, and Lecturer on Dental Surgery at, the Middlesex Hospital; Dental Surgeon to, and Lecturer on Dental Surgery and Pathology at, the Dental Hospital of London; 17, George street, Hanover square.
- 1877 BENNETT, WILLIAM HENRY, Surgeon to St. George's Hospital; 1, Chesterfield street, Mayfair. C. 1893-4. *Referee*, 1892-93. *Trans.* 4.
- †1845 BERRY, EDWARD UNWIN, 17, Sherrieff road, West Hampstead.
- 1885 BERRY, JAMES, B.S., Demonstrator of Anatomy, St. Bartholomew's Hospital; Surgeon to, and Lecturer on Clinical Surgery at, the Royal Free Hospital; 60, Welbeck street, Cavendish square.
- 1893 BIDWELL, LEONARD A., Senior Assistant Surgeon to the West London Hospital; 59, Wimpole street.
- †1856 BIRD, WILLIAM, Consulting Surgeon to the West London Hospital; Bute House, Hammersmith.
- †1851 BIRKETT, JOHN, F.L.S., Consulting Surgeon to Guy's Hospital; Corresponding Member of the Société de Chirurgie of Paris; Inspector of Anatomy for the Provinces in England and Wales; 62, Green street, Grosvenor square. L. 1856-7. S. 1863-5. C. 1867-8. T. 1870-78. V.P. 1879-80. *Referee*, 1851-5, 1866, 1869. *Sci. Com.* 1863. *Lib. Com.* 1852. *Trans.* 8.
- 1881 BISS, CECIL YATES, M.D., Senior Assistant Physician to, and Lecturer on Pharmacology and Therapeutics at, the Middlesex Hospital; Physician to the Hospital for Consumption, Brompton; 135, Harley street, Cavendish square. *Trans.* 2.

*Elected*

- 1865 BLANDFORD, GEORGE FIELDING, M.D., late Lecturer on Psychological Medicine at St. George's Hospital; 48, Wimpole street, Cavendish square. C. 1883-4.
- 1891 BOKENHAM, THOMAS JESSOPP, 10, Devonshire street, Portland place.
- 1890 BOSTOCK, R. ASHTON, Surgeon, Scots Guards, 73, Onslow gardens, Brompton.
- 1882 BOWLBY, ANTHONY ALFRED, Assistant Surgeon to St. Bartholomew's Hospital; 24, Manchester square. *Trans.* 6.
- \*1870 BOWLES, ROBERT LEAMON, M.D., 16, Upper Brook street, Grosvenor square. *Trans.* 1.
- 1894 BOWMAN, HENRY MOORE, M.D., 21, Welbeck street, Cavendish square.
- 1886 BOXALL, ROBERT, M.D., Assistant Obstetric Physician to, and Lecturer on Practical Midwifery at, the Middlesex Hospital; 29, Weymouth street, Portland place.
- 1884 BOYD, STANLEY, M.B., Surgeon to, and Lecturer on Anatomy at, the Charing Cross Hospital; Surgeon to the Paddington Green Children's Hospital; 134, Harley street, Cavendish square. *Referee*, 1895. *Trans.* 1.
- 1890 BRADFORD, JOHN ROSE, M.D., D.Sc., F.R.S., Assistant Physician to University College Hospital; 52, Upper Berkeley street, Portman square.
- 1883 BRADSHAW, JAMES DIXON, M.B., Savile Club, Piccadilly, W.
- 1890 BRINTON, ROLAND DANVERS, M.D., 8, Queen's Gate terrace.
- 1868 BROADBENT, SIR WILLIAM HENRY, Bart., M.D., Physician to, and Lecturer on Clinical Medicine at, St. Mary's Hospital; Consulting Physician to the London Fever Hospital; Physician in Ordinary to H.R.H. the Prince of Wales; 84, Brook street, Grosvenor square. C. 1885. *Referee*, 1881-4, 1891—. *Trans.* 5.

*Elected*

- †1851 **BRODHURST, BERNARD EDWARD**, Surgeon to the Royal Orthopædic Hospital and to the Royal Hospital for Incurables; Consulting Surgeon, Belgrave Hospital for Children; Corresponding Member Société de Chirurgie, Paris, and of the Academy of Sciences, Rome; 20, Grosvenor street. C. 1868-9. *Lib. Com.* 1862-3. *Trans.* 2. *Pro.* 1.
- 1872 **BRODIE, GEORGE BERNARD, M.D.**, Consulting Physician-Accoucheur to Queen Charlotte's Hospital; 8, Chesterfield street, Mayfair. *Trans.* 1.
- 1891 **BRODIE, CHARLES GORDON**, Assistant Surgeon, North-West London Hospital; 30, Harley street, Cavendish square.
- 1880 **BROWNE, JAMES WILLIAM, M.B.**, 7, Norland place, Holland Park.
- 1881 **BROWNE, OSWALD AUCHINLECK, M.A., M.B.**, Physician to the Royal Hospital for Diseases of the Chest; 43, Bedford square.
- 1874 **BRUCE, JOHN MITCHELL, M.D.**, *Hon. Secretary*; Physician to, and Lecturer on Medicine at, the Charing Cross Hospital; Physician to the Hospital for Consumption, Brompton; 23, Harley street. C. 1892. S. 1894—. *Sci. Com.* 1889—. *Referee*, 1886-91. *Lib. Com.* 1888-91. *Trans.* 3.
- 1871 **BRUNTON, THOMAS LAUDER, M.D., D.Sc., LL.D., F.R.S.**, Assistant Physician to, and Lecturer on *Materia Medica* and Therapeutics at, St. Bartholomew's Hospital; 10, Stratford place, Oxford street. C. 1888-9. *Referee*, 1880-87. *Lib. Com.* 1882-7. *Trans.* 1.
- 1860 **BRYANT, THOMAS**, Consulting Surgeon to Guy's Hospital; Member of the Société de Chirurgie, Paris; 65, Grosvenor street, Grosvenor square. C. 1873-4. V. P. 1885-6. *Sci. Com.* 1863. *Referee*, 1882-4. *Lib. Com.* 1868-71. *Trans.* 15. *Pro.* 1.

*Elected*

- 1889 BULL, WILLIAM CHARLES, M.B., Aural Surgeon to, and Lecturer on Aural Surgery at, St. George's Hospital; 35, Clarges street, Piccadilly.
- 1893 BURGHARD, FRÉDÉRIC FRANÇOIS, M.D., M.S., Assistant Surgeon to King's College Hospital; 46, Weymouth street, Portland place.
- 1885 BUTLER-SMYTHE, ALBERT CHARLES, Senior Surgeon to the Grosvenor Hospital for Women and Children; 76, Brook street, Grosvenor square.
- 1873 BUTLIN, HENRY TEENTHAM, D.C.L., Surgeon to St. Bartholomew's Hospital; 82, Harley street, Cavendish square. C. 1887-8. *Referee*, 1893—. *Trans.* 4.
- 1883 BUXTON, DUDLEY WILMOT, M.D., B.S., Administrator, and Teacher of the Use, of Anæsthetics, in University College Hospital; Anæsthetist to the National Hospital for the Paralysed and Epileptic, Queen's square, and to the London Dental Hospital; 82, Mortimer street, Cavendish square.
- 1868 BUZZARD, THOMAS, M.D., Physician to the National Hospital for the Paralysed and Epileptic; 74, Grosvenor street, Grosvenor square. C. 1885-6. *Referee*, 1887—.
- 1890 CAGNEY, JAMES, M.A., M.D., in charge of Electrical Department, St. Mary's Hospital; Physician to the Hospital for Epilepsy and Paralysis, Regent's Park; 93, Wimpole street, Cavendish square. *Trans.* 1.
- 1885 CAHILL, JOHN, 12, Seville street, Lowndes square.
- 1893 CALEY, HENRY ALBERT, M.D., Medical Registrar and Joint Medical Tutor, St. Mary's Hospital; 24, Upper Berkeley street, Portman square.
- 1887 CALVERT, JAMES, M.D., 36, Queen Anne street, Cavendish square.
- 1891 CAMPBELL, HENRY JOHNSTONE, M.D., Senior Demonstrator of Biology and Demonstrator of Physiology, Guy's Hospital; Assistant Physician, East London Children's Hospital; 54, Welbeck street.

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- 1888 CARLESS, ALBERT, M.S., Assistant Surgeon to King's College Hospital; 10, Welbeck street.
- 1875 CARTER, CHARLES HENRY, M.D., Physician to the Hospital for Women, Soho square; 45, Great Cumberland place, Hyde Park.
- 1853 CARTER, ROBERT BRUDENELL, Consulting Ophthalmic Surgeon to St. George's Hospital; 31, Harley street, Cavendish square. *Trans.* 1.
- 1888 CAUTLEY, EDMUND, M.D., B.C., 15, Upper Brook street.
- 1868 CAVAFY, JOHN, M.D., Physician to St. George's Hospital; 2, Upper Berkeley street, Portman square. C. 1887. *Lib. Com.* 1888—. *Trans.* 1.
- 1871 CAYLEY, WILLIAM, M.D., Physician to, and Lecturer on the Principles and Practice of Medicine at, the Middlesex Hospital, Consulting Physician to the London Fever Hospital, and to the North-Eastern Hospital for Children; 27, Wimpole street, Cavendish square. C. 1888. *Referee*, 1886-7. *Lib. Com.* 1886-7. *Trans.* 2.
- 1879 CHAMPNEYS, FRANCIS HENRY, M.A., M.D., Physician Accoucheur and Lecturer on Obstetric Medicine at St. Bartholomew's Hospital; 42, Upper Brook street, Grosvenor square. *Referee*, 1891—. *Lib. Com.* 1885—. *Trans.* 8.
- 1868 CHEADLE, WALTER BUTLER, M.D., *Trustee*; Physician to, and Lecturer on Medicine at, St. Mary's Hospital; Consulting Physician to the Hospital for Sick Children; 19, Portman street, Portman square. S. 1886-8. C. 1890-91. *Sci. Com.* 1889—. *Bldg. Com.* 1889-92. *Referee*, 1885. *Trans.* 1.
- 1879 CHEYNE, WILLIAM WATSON, M.B., F.R.S., Surgeon to King's College Hospital, and Professor of Surgery in King's College, London; 75, Harley street, Cavendish square. *Referee*, 1894—. *Lib. Com.* 1886-8, 1891—. *Trans.* 1.

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- 1866 CHURCH, WILLIAM SELBY, M.D., *Hon. Treasurer*, Physician to, and Lecturer on Clinical Medicine at, St. Bartholomew's Hospital; 130, Harley street, Cavendish square. C. 1885-6. V.P. 1892-4. T. 1894—. *Referee*, 1874-81.
- 1879 CLARK, ANDREW, Surgeon to, and Lecturer on Practical Surgery at, the Middlesex Hospital; 71, Harley street, Cavendish square.
- 1882 CLARKE, ERNEST, M.D., B.S., Surgeon to the Central London Ophthalmic Hospital; Ophthalmic Surgeon to the Miller Hospital; 112, Harley street.
- 1890 CLARKE, JAMES JACKSON, M.B., Curator of the Museum and Pathologist to St. Mary's Hospital, 9, Old Cavendish street, W.
- †1848 CLARKE, JOHN, M.D., 42, Hertford street, Mayfair. C. 1866.
- 1881 CLARKE, W. BRUCE, M.B., Assistant Surgeon to, and Lecturer on Anatomy at, St. Bartholomew's Hospital; Surgeon to the West London Hospital; 51, Harley street, Cavendish square. *Trans.* 1.
- †1879 CLUTTON, HENRY HUGH, M.A., M.B., Surgeon to, and Lecturer on Surgery at, St. Thomas's Hospital; Surgeon to the Victoria Hospital for Children; 2, Portland place. *Trans.* 1.
- 1888 COCK, FREDERICK WILLIAM, M.D., 1, Porchester Houses, Porchester square.
- 1865 COOPER, ALFRED, Consulting Surgeon to the West London Hospital; Senior Surgeon to St. Mark's Hospital; 9, Henrietta street, Cavendish square.
- 1889 COSENS, CHARLES HENRY, 49, Oxford terrace, Hyde Park,
- 1892 COTTERELL, EDWARD, Surgeon for Out-patients, London Lock Hospital; Surgeon to the Cancer Hospital; Surgeon to the West-End Hospital for Epilepsy and Diseases of the Nervous System; 5, West Halkin street, Belgrave square. *Trans.* 1.

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- †1860 COUPER, JOHN, Surgeon to the Royal London Ophthalmic Hospital, and Consulting Surgeon to the London Hospital; 80, Grosvenor street. C. 1876. *Referee*, 1882-3.
- 1877 COUPLAND, SIDNEY, M.D., Physician to, and Joint Lecturer on Practical Medicine at, the Middlesex Hospital; 16, Queen Anne street, Cavendish square. C. 1893-4. *Referee*, 1892-3. *Ho. Com.*, 1895.
- †1862 COWELL, GEORGE, Surgeon to, and Lecturer on Surgery at, the Westminster Hospital; Surgeon to the Royal Westminster Ophthalmic Hospital; Consulting Surgeon to the Victoria Hospital for Children; 7, Henrietta street, Cavendish square. C. 1882-3.
- 1868 CRAWFORD, SIR THOMAS, K.C.B., M.D., M.Ch., LL.D., Hon. Surgeon to the Queen; Director-General, Army Medical Department (Retired); 5, St. John's Park, Blackheath. C. 1887.
- 1878 CRICHTON-BROWNE, SIR JAMES, M.D., LL.D., F.R.S., Lord Chancellor's Visitor in Lunacy; 61, Carlisle place Mansions, Victoria street.
- 1874 CRIPPS, WILLIAM HARRISON, Surgeon to St. Bartholomew's Hospital; 2, Stratford place, Oxford street. C. 1890-91. *Trans.* 1.
- 1892 CROCKER, HENRY RADCLIFFE, M.D., Physician to the Skin Department, University College Hospital; late Physician to the East London Hospital for Children; 121, Harley street, Cavendish square. *Trans.* 3.
- 1868 CROFT, JOHN, Consulting Surgeon to St. Thomas's Hospital; 6, Mansfield street, Cavendish square. C. 1884. V.P. 1890. *Referee*, 1885-88. *Lib. Com.* 1877-8. *Trans.* 2.
- 1890 CROWLE, THOMAS HENRY RICKARD, 3, Campden Hill road, Kensington.
- 1888 CULLINGWORTH, CHARLES JAMES, M.D., Obstetric Physician and Lecturer on Midwifery at St. Thomas's Hospital; 46, Brook street, Grosvenor square.



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- 1879 CUMBERBATCH, A. ELKIN, Aural Surgeon to St. Bartholomew's Hospital, and to the National Hospital for the Paralysed and Epileptic; 80, Portland place, W.
- 1873 CURNOW, JOHN, M.D., Professor of Anatomy in King's College, London, and Physician to King's College Hospital; Senior Physician to the Seamen's Hospital; 11, Wimpole street, Cavendish square. *Referee*, 1884—.
- 1886 DAKIN, WILLIAM RADFORD, M.D., Obstetric Physician to St. George's Hospital; 57, Welbeck street, Cavendish square.
- 1872 DALBY, SIR WILLIAM BARTLETT, M.B., Consulting Aural Surgeon to St. George's Hospital; 18, Savile row. *Trans.* 3.
- 1891 DALTON, NORMAN, M.D., Physician to King's College Hospital; 4, Mansfield street, Cavendish square.
- 1876 DAVIES-COLLEY, J. NEVILLE C., M.C., Surgeon to, and Lecturer on Surgery at, Guy's Hospital; 36, Harley street, Cavendish square. C. 1892-3. *Referee*, 1890-91. *Trans.* 3.
- 1889 DEAN, HENRY PERCY, M.S., Assistant Surgeon to the London Hospital; 84, Wimpole street, Cavendish square.
- 1878 DENT, CLINTON THOMAS, Surgeon to, and Lecturer on Practical Surgery at, St. George's Hospital; 61, Brook street. C. 1890. *Bldg. Com.* 1890-2. *Referee*, 1892—. *Trans.* 4.
- 1891 DE SANTI, PHILIP ROBERT WILLIAM, 37, Queen Anne street, Cavendish square.
- 1894 DICKINSON, THOMAS VINCENT, M.D., 33, Sloane street, S.W.

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- †1859 DICKINSON, WILLIAM HOWSHIP, M.D., Consulting Physician to St. George's Hospital, and Consulting Physician to the Hospital for Sick Children; Honorary Fellow of Caius College, Cambridge; 9, Chesterfield street, Mayfair. C. 1874-5. V.P. 1887. *Referee*, 1869-73. 1882-6. *Sci. Com.* 1867, 1879, 1889—. *Trans.* 15.
- †1891 DICKINSON, WILLIAM LEE, M.D., Assistant Physician to St. George's Hospital; 9, Chesterfield street, Mayfair.
- 1889 DODD, HENRY WORK, Assistant Surgeon to the Royal Free Hospital, and to the Royal Westminster Ophthalmic Hospital; Ophthalmic Surgeon to the West-End Hospital for Nervous Diseases; 136, Harley street, Cavendish square.
- 1888 DONELAN, JAMES, M.B., M.C., Physician to the Italian Hospital, Queen square; 2, Upper Wimpole street, Cavendish square.
- 1879 DONKIN, HORATIO BRYAN, M.D.Oxon., Physician to the Westminster Hospital; Physician to the East London Hospital for Children; 108, Harley street, Cavendish square.
- 1877 DORAN, ALBAN HENRY GRIFFITHS, Surgeon to the Samaritan Free Hospital; 9, Granville place, Portman square. C. 1893-94. *Lib. Com.* 1891-3. *Trans.* 4.
- 1891 DOVE, PERCY W., "Carshalton," Stapleton Hall Road, Stroud Green, N.
- 1879 DREWITT, F. G. DAWTREY, M.D., Physician to the West London Hospital and to the Victoria Hospital for Children; 2, Manchester square.
- †1865 DUCKWORTH, SIR DYCE, M.D., LL.D., Hon. Physician to H.R.H. the Prince of Wales; Physician to, and Lecturer on Medicine at, St. Bartholomew's Hospital; 11, Grafton street, Bond street. C. 1883-4. *Referee*, 1885—. *Trans.* 2.
- 1876 DUDLEY, WILLIAM LEWIS, M.D., Senior Physician to the City Dispensary; 149, Cromwell road, South Kensington.

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- 1874 DUFFIN, ALFRED BAYNARD, M.D., Professor of Pathological Anatomy in King's College, London, and Physician to King's College Hospital; 18, Devonshire street, Portland place. C. 1893-94.
- 1871 DUKE, BENJAMIN, Windmill House, Clapham Common.
- 1880 DUNBAR, JAMES JOHN MACWHIRTER, M.D., Hedingham House, Clapham Common.
- 1884 DUNCAN, WILLIAM, M.D., Obstetric Physician to, and Lecturer on Midwifery at, the Middlesex Hospital; 6, Harley street, Cavendish square.
- 1887 DUNN, HUGH PERCY, Assistant Ophthalmic Surgeon to the West London Hospital; 54, Wimpole street, Cavendish square.
- 1874 DURHAM, FREDERIC, M.B., Senior Surgeon to the North-West London Hospital; late Surgical Registrar to Guy's Hospital; 82, Brook street, Grosvenor square.
- 1893 DURHAM, HERBERT EDWARD, M.B., 82, Brook street, Grosvenor Square. *Trans.* 1.
- 1868 EASTES, GEORGE, M.B. Lond., 35, Gloucester place, Hyde Park. C. 1892-3.
- 1888 ECCLES, ARTHUR SYMONS, M.B., C.M., 23, Hertford street, Mayfair.
- 1893 ECCLES, WILLIAM MCADAM, M.D., 10, Welbeck street.
- 1891 EDDOWES, ALFRED, M.D., 25, Old Burlington street.
- 1883 EDMUNDS, WALTER, M.C., 75, Lambeth Palace road, Albert Embankment. *Trans.* 2.
- 1884 EDWARDS, FREDERICK SWINFORD, Surgeon to the West London Hospital, and to St. Peter's Hospital for Stone; 55, Harley street, Cavendish square.
- †1842 ERICHSEN, SIR JOHN ERIC, Bart., LL.D., F.R.S., M.Ch., Surgeon Extraordinary to H.M. the Queen; President of, and Emeritus Professor of Surgery in, University College, London, and Consulting Surgeon to University College Hospital; 6, Cavendish place, Cavendish square. C. 1855-6. V.P. 1868. P. 1879-80. *Referee*, 1866-8, 1884-89. *Lib. Com.* 1844-7, 1854. *Trans.* 2.

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- 1879 EVE, FREDERIC S., Surgeon to the London Hospital; Surgeon to the Evelina Hospital for Sick Children; 125, Harley street, Cavendish square. *Trans.* 2.
- 1877 EWART, WILLIAM, M.D., Physician to St. George's Hospital; 33, Curzon street, Mayfair. C. 1895—. *Sci. Com.* 1889—. *Trans.* 1.
- 1872 FAYRER, SIR JOSEPH, K.C.S.I., LL.D., M.D., F.R.S., Surgeon-General; Honorary Physician to H.M. the Queen, (Military) to H.R.H. the Prince of Wales, and Physician to H.R.H. the Duke of Edinburgh; Physician to the Secretary of State for India in Council, and President of the Medical Board at the India Office; 53, Wimpole street, Cavendish square. C. 1888. *Referee*, 1881-7.
- 1863 FENWICK, SAMUEL, M.D., Physician to the London Hospital; 29, Harley street, Cavendish square. C. 1880. *Referee*, 1882—. *Trans.* 4.
- 1880 FERRIER, DAVID, M.D., LL.D., F.R.S., Professor of Neuro-pathology in King's College, London, and Physician to King's College Hospital; Physician to the National Hospital for the Paralysed and Epileptic; 34, Cavendish square. *Referee*, 1891—. *Trans.* 2.
- 1889 FIELD, GEORGE P., Aural Surgeon to, and Lecturer on Aural Surgery at, St. Mary's Hospital, and Dean of the Medical School; 34, Wimpole street, Cavendish square.
- 1866 FITZ-PATRICK, THOMAS, A.M., M.D., 30, Sussex gardens, Hyde Park. C. 1894-5.
- 1891 FLETCHER, HERBERT MORLEY, M.D., 98, Harley street, Cavendish square.
- 1892 FORSBROOK, WILLIAM HENRY RUSSELL, M.D., 139, Buckingham Palace road.
- 1882 FOWLER, JAMES KINGSTON, M.A., M.D., Physician to, and Lecturer on Pathological Anatomy at, the Middlesex Hospital; Physician to the Hospital for Consumption, Brompton; 35, Clarges street, Piccadilly.

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- 1880 FOX, THOMAS COLCOTT, B.A., M.B., Physician for Skin Diseases to the Westminster Hospital, and to the Skin Department of the Paddington Green Hospital for Children; late Physician to the Victoria Hospital for Children; 14, Harley street, Cavendish square. *Trans.* 1.
- 1884 FULLER, CHARLES CHINNER, 10, St. Andrew's place, Regent's Park.
- 1883 FULLER, HENRY ROXBURGH, M.D., 45, Curzon street, Mayfair.
- 1894 FURNIVALL, PERCY, 34, Adelaide road, South Hampstead.
- †1874 GALABIN, ALFRED LEWIS, M.A., M.D., Obstetric Physician to, and Lecturer on Midwifery and the Diseases of Women at, Guy's Hospital; 49, Wimpole st., Cavendish square. C. 1892. *Referee*, 1882-91. *Lib. Com.* 1883-4. *Trans.* 2.
- 1883 GALTON, JOHN CHARLES, M.A., F.L.S., 10, Upper Cheyne row, Chelsea.
- 1865 GANT, FREDERICK JAMES, Consulting Surgeon to the Royal Free Hospital; 16, Connaught square, Hyde Park. C. 1880-81. *Referee*, 1886—. *Lib. Com.* 1882-5. *Trans.* 3.
- †1854 GARROD, SIR ALFRED BARING, M.D., F.R.S., Physician Extraordinary to H.M. the Queen; Consulting Physician to King's College Hospital; 10, Harley street, Cavendish square. C. 1867. V.P. 1880-81. *Referee*, 1855-65. *Trans.* 8.
- 1886 GARROD, ARCHIBALD EDWARD, M.A., M.D., Assistant Physician to the West London Hospital; 9, Chandos street, Cavendish square. *Sci. Com.* 1889—. *Trans.* 5.
- 1887 GAY, JOHN, 119, Upper Richmond road, Putney.
- 1866 GEE, SAMUEL JONES, M.D., *Hon. Librarian; Chairman of Trustees for Debenture-holders*; Physician to St. Bartholomew's Hospital; 31, Upper Brook street, Grosvenor square. C. 1883-4. L. (June) 1887—. *Sci. Com.* 1879. *Bldg. Com.* 1889-92. *Referee*, 1885-7. *Lib. Com.* 1871-6. *Trans.* 1.

*Elected*

- 1885 GELL, HENRY WILLINGHAM, M.B., 36, Hyde Park square.
- 1878 GEEVIS, HENRY, M.D., Consulting Obstetric Physician to St. Thomas's Hospital; Consulting Physician to the Royal Maternity Charity; 40, Harley street, Cavendish square. *Referee*, 1884—. *Trans.* 1.
- 1880 GIBBONS, ROBERT ALEXANDER, M.D., Physician to the Grosvenor Hospital for Women and Children; 29, Cadogan place.
- 1893 GILES, ARTHUR EDWARD, M.D., B.Sc., 57, Queen Anne street.
- 1894 GILL, RICHARD, 72, Wimpole street.
- 1877 GODLEE, RICKMAN JOHN, M.S., *Hon. Librarian*; Surgeon to University College Hospital, and Professor of Clinical Surgery in University College, London; Surgeon to the Hospital for Consumption, Brompton; Consulting Surgeon to the North-Eastern Hospital for Children; 19, Wimpole street, Cavendish square. *S.* 1892-4. *Hon. L.* 1895. *Referee*, 1886-91 *Trans.* 8.
- †1870 GODSON, CLEMENT, M.D., Consulting Physician to the City of London Lying-in Hospital; 9, Grosvenor street, Grosvenor square.
- 1886 GOLDING-BIRD, CUTHBERT HILTON, M.B., Surgeon to, and Lecturer on Physiology at, Guy's Hospital; 12, Queen Anne street, Cavendish square. *Trans.* 1.
- 1883 GOODHART, JAMES FREDERIC, M.D., Physician to Guy's Hospital; Consulting Physician to the Evelina Hospital for Sick Children; 25, Portland place. *Lib. Com.* 1893—.
- 1889 GOODSALL, DAVID HENRY, Surgeon to the Metropolitan Hospital; Surgeon to St. Mark's Hospital; 17, Devonshire place, Upper Wimpole street.
- 1893 GORDON, WILLIAM, M.B., M.C.
- 1895 GOSSAGE, ALFRED MILNE, M.B., 54, Upper Berkeley street.

*Elected*

- 1877 GOULD, ALFRED PEARCE, M.S., Surgeon to the Middlesex Hospital; 10, Queen Anne street, Cavendish square. C. 1892-3. *Referee*, 1895—. *Ho. Com.* 1891—. *Trans.* 2.
- 1891 GOW, WILLIAM J., M.D., Assistant Obstetric Physician to St. Mary's Hospital; Obstetric Physician to the Royal Hospital for Women and Children; Physician to Out-Patients, Queen Charlotte's Lying-in Hospital; 27, Weymouth street, Portland place.
- 1873 GOWERS, WILLIAM RICHARD, M.D., F.R.S., Consulting Physician to University College Hospital; Physician to the National Hospital for the Paralysed and Epileptic; 50, Queen Anne street, Cavendish square. C. 1891. *Referee* 1888-90. *Lib. Com.* 1884-6. *Trans.* 7.
- †1851 GOWLLAND, PETER YEAMES, late Senior Surgeon to St. Mark's Hospital; Brigade Surgeon Hon. Artillery Company; 82, Gloucester terrace, Hyde park.
- 1892 GRANT, J. DUNDAS, M.A., M.D., 8, Upper Wimpole street, Cavendish square.
- 1868 GREEN, T. HENRY, M.D., Physician to the Charing Cross Hospital, and to the Hospital for Consumption, Brompton; 74, Wimpole street, Cavendish square. C. 1886. *Referee*, 1882-5.
- 1885 GRIFFITH, WALTER SPENCER ANDERSON, M.D., Assistant Physician-Accoucheur, St Bartholomew's Hospital; Physician to Queen Charlotte's Lying-in Hospital; 96, Harley street, Cavendish square.
- 1868 GRIGG, WILLIAM CHAPMAN, M.D., Obstetric Physician to the Out-patients at the Westminster Hospital; Physician to the In-Patients, Queen Charlotte's Lying-in Hospital; Joint Lecturer on Forensic Medicine at the Westminster Hospital Medical School; 27, Curzon street, Mayfair.
- 1889 GUBB, ALFRED S, M.D. Paris; 29, Gower street.

*Elected*

- 1883 GUNN, ROBERT MARCUS, M.A., M.B., Surgeon to the Royal London Ophthalmic Hospital, Moorfields; Ophthalmic Surgeon to the National Hospital for the Paralyaed and Epileptic; Assistant Ophthalmic Surgeon to University College Hospital; 54, Queen Anne street, Cavendish square.
- 1890 GUTHRIE, LEONARD GEORGE, M.D., B.Ch., Physician to the Regent's Park Hospital for Epilepsy and Paralysis; Assistant Physician to the North-West London Hospital; Assistant Physician to the Children's Hospital, Paddington Green; 15, Upper Berkeley street, Portman square.
- 1886 HABERSHON, SAMUEL HERBERT, M.D., Assistant Physician to the Hospital for Consumption, Brompton; 70, Brook street, Grosvenor square.
- 1885 HAIG, ALEXANDER, M.D., Physician to the Metropolitan Hospital, and to the Royal Hospital for Children and Women; 7, Brook street, Grosvenor square. *Trans.* 6.
- 1890 HALE, CHARLES DOUGLAS BOWDICH, M.D., 3, Sussex place, Hyde Park.
- 1881 HALL, FRANCIS DE HAVILLAND, M.D., Physician to, and Physician to the Throat Department at, the Westminster Hospital; Physician to St. Mark's Hospital; 47, Wimpole street, Cavendish square. *Referee*, 1893—.
- 1891 HAMER, WILLIAM HEATON, M.D., 73, Dartmouth Park Hill, Highgate.
- 1889 HANDFIELD-JONES, MONTAGU, M.D., Obstetric Physician to, and Lecturer on Midwifery and Diseases of Women at, St. Mary's Hospital; Physician to the British Lying-in Hospital; 35, Cavendish square.
- †1856 HARE, CHARLES JOHN, M.D., Consulting Physician to University College Hospital; Berkeley House, 15, Manchester square. C. 1873-4. T. 1887-94. V.P. 1894-5. *Bldg. Com.* 1889-92.
- †1857 HARLEY, GEORGE, M.D., F.R.S. 25, Harley street, Cavendish square. C. 1871-2. V.P. 1891-2. *Referee*, 1865-70, 1873-6. *Sci. Com.* 1862-3, 1866, 1889—. *Trans.* 1.



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- †1864 HARLEY, JOHN, M.D., F.L.S., Physician to, and Lecturer on Clinical Medicine at, St. Thomas's Hospital; 9, Stratford place, Oxford street. S. 1875-7. C. 1879-80. V.P. 1895—. *Referee*, 1871-4, 1882-95. *Sci. Com.* 1879. *Trans.* 10.
- 1893 HARLEY, VAUGHAN, M.D., 25, Harley street, W.
- 1892 HAROLD, JOHN, 91, Harley street, Cavendish square.
- 1880 HARRIS, VINCENT DORMER, M.D., Physician to the City of London Hospital for Diseases of the Chest, Victoria Park; 31, Wimpole street, Cavendish square.
- 1870 HARRISON, REGINALD, 6, Lower Berkeley Street, Portman square. C. 1894-5. *Trans.* 2.
- 1870 HAWARD, J. WARRINGTON, *Hon. Treasurer*; Surgeon to, and Lecturer on Clinical Surgery at, St. George's Hospital; 16, Savile row, Burlington Gardens. C. 1885. S. 1888-91. V.P. 1894-5 (June) 1895—. *Lib. Com.* 1881-4. *Sci. Com.* 1889-91. *Bldg. Com.* (Sec.) 1889-92. *Ho. Com.* 1892—. *Trans.* 2.
- 1891 HAWKINS, HERBERT PENNELL, M.B., B.C., Assistant Physician to St. Thomas's Hospital; 109, Harley street, Cavendish square.
- 1875 HAYES, THOMAS CRAWFORD, M.A., M.D., Physician-Accoucheur and Physician for Diseases of Women and Children to King's College Hospital, and Lecturer on Practical Obstetrics in King's College; Physician for Diseases of Women to the Royal Free Hospital; 17, Clarges street, Piccadilly.
- 1860 HAYWARD, HENRY HOWARD, Consulting Surgeon Dentist to St. Mary's Hospital; 38, Harley street, Cavendish square. C. 1878-9.
- 1891 HAYWARD, JOHN ARTHUR, M.D., 58, Brook street.
- †1865 HEATH, CHRISTOPHER, Holme Professor of Clinical Surgery in University College, London; and Surgeon to University College Hospital; 36, Cavendish square. C. 1880. V.P. 1889. *Lib. Com.* 1870-3. *Trans.* 3.

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- 1882 HENSLEY, PHILIP JOHN, M.D., Physician to, and Lecturer on Forensic Medicine at, St. Bartholomew's Hospital; 4, Henrietta street, Cavendish square.
- 1877 HERMAN, GEORGE ERNEST, M.B., Obstetric Physician to, and Lecturer on Midwifery at, the London Hospital; 20, Harley street, Cavendish square. *Referee*, 1892—. *Trans.* 1.
- 1877 HERON, GEORGE ALLAN, M.D., Physician to the City of London Hospital for Diseases of the Chest, Victoria Park; 57, Harley street, Cavendish square.
- 1891 HERRING, HERBERT T., M.B., B.S., 50, Harley street, Cavendish square.
- 1883 HERRINGHAM, WILMOT PARKER, M.D., Medical Registrar, St. Bartholomew's Hospital; 13, Upper Wimpole street, Cavendish square. *Trans.* 1.
- 1893 HERSCHELL, GEORGE, M.D., 25, Queen Anne street, Cavendish square.
- 1887 HEWITT, FREDERIC WILLIAM, M.D., Anæsthetist to, and Instructor in Anæsthetics at, the London Hospital; Chloroformist to, and Lecturer on Anæsthetics at, Charing Cross Hospital; Anæsthetist at the Dental Hospital of London; 10, George street, Hanover square. *Trans.* 1.
- 1873 HIGGINS, CHARLES, Ophthalmic Surgeon to, and Lecturer on Ophthalmic Surgery at, Guy's Hospital; 38, Brook street, Grosvenor square. C. 1894-5. *Trans.* 2.
- 1890 HILL, G. WILLIAM, M.D., B.Sc., 24, Wimpole street, Cavendish square.
- †1856 HOLMES, TIMOTHY, M.A., Consulting Surgeon to St. George's Hospital; Corresponding Member of the Société de Chirurgie, Paris; 6, Sussex place, Hyde Park. C. 1869-70. L. 1873-7. S. 1878-80. V.P. 1881-2. T. 1885-7. P. 1890-92. *Bldg. Com.* (*Chairman*) 1889-92. *Referee*, 1866-8, 1872, 1883-4. *Sci. Com.* 1867. *Lib. Com.* 1863-5, 1892-5. *Ho. Com.* 1892—. *Trans.* 8.
- 1878 HOOD, DONALD WILLIAM CHARLES, M.D., Senior Physician to the West London Hospital; 43, Green street, Park lane.

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- 1883 HORSLEY, VICTOR ALEXANDER HADEN, F.R.S., Surgeon to University College Hospital, Surgeon to the National Hospital for the Paralysed and Epileptic; Professor of Pathology in University College, London; 25, Cavendish square. *Trans.* 1.
- 1892 HOWARD, R. J. BLISS, M.D., 31, Queen Anne street, Cavendish square.
- 1874 HOWSE, HENRY GREENWAY, M.S., Surgeon to, and Lecturer on Surgery at, Guy's Hospital; Consulting Surgeon to the Evelina Hospital for Sick Children; 59, Brook street, Grosvenor square. C. 1890. *Sci. Com.* 1879. *Referee*, 1887-89. *Trans.* 3.
- 1886 HUDSON, CHARLES ELLIOTT LEOPOLD BARTON, Assistant Surgeon and Surgeon to Aural Department, Middlesex Hospital; Surgeon to Aural Department to the Hospital for Sick Children, Great Ormond Street; 16, Harley street, Cavendish square.
- 1889 HUMPHERY, FRANCIS WILLIAM, M.A., M.B., 63, Prince's gate.
- 1889 HUNTER, WILLIAM, M.D., Senior Assistant Physician to the London Fever Hospital; 54, Harley street.
- 1873 HUNTER, SIR W. GUYER, M.D., K.C.M.G., Hon. Surgeon to H.M. the Queen; formerly Principal of, and Professor of Medicine in, Grant Medical College, and Vice-Chancellor of the University, Bombay; Surgeon-General (Retired) Bombay Army; Consulting Physician to Charing Cross Hospital; 21, Norfolk crescent, Hyde Park.
- †1856 HUTCHINSON, JONATHAN, F.R.S., *President*; Consulting Surgeon to, and Emeritus Professor of Surgery at, the London Hospital; Consulting Surgeon to the Royal London Ophthalmic Hospital, Moorfields; and Senior Surgeon to the Hospital for Diseases of the Skin; 15, Cavendish square. C. 1870. V.P. 1882. P. 1894-5. *Referee*, 1876-81, 1883-94. *Lib. Com.* 1864-5. *Trans.* 14. *Pro.* 2.

*Elected*

- 1888 HUTCHINSON, JONATHAN, Jun., Assistant Surgeon to the London Hospital; 1, Park crescent. *Trans.* 1.
- 1871 JACKSON, J. HUGHLINGS, M.D., F.R.S., Consulting Physician to the London Hospital; Physician to the National Hospital for the Paralysed and Epileptic; 3, Manchester square. C. 1889.
- †1841 JACKSON, PAUL, 51, Wellington road, St. John's Wood. C. 1862.
- 1883 JACOBSON, WALTER HAMILTON ACLAND, M.A., M.B., M.Ch., Assistant Surgeon and Lecturer on Anatomy to Guy's Hospital; Surgeon to the Royal Hospital for Children and Women; 66, Great Cumberland place, Hyde Park. *Referee*, 1895—. *Trans.* 2.
- 1892 JAMES, EDWIN MATTHEWS, Belgrave Mansions, Grosvenor gardens.
- 1884 JESSETT, FREDERIC BOWREMAN, Surgeon to the Cancer Hospital, Brompton; 1, Buckingham Palace Mansions.
- 1883 JESSOP, WALTER H. H., M.B., Ophthalmic Surgeon to St. Bartholomew's Hospital; 73, Harley street.
- †1847 JOHNSON, SIR GEORGE, M.D., F.R.S., Physician Extraordinary to H.M. the Queen; Consulting Physician to King's College Hospital; Emeritus Professor of Clinical Medicine, Fellow, and Member of the Council, King's College, London; Member of the Senate of the University of London; 11, Savile row, Burlington gardens. C. 1862-3. V.P. 1870. P. 1884-5. L. 1878-80. *Referee*, 1853-61, 1864-9. *Lib. Com.* 1860-1. *Trans.* 10. *Pro.* 1.
- 1881 JOHNSON, GEORGE LINDSAY, M.A., M.D., Cortina, Netherhall gardens, South Hampstead, and 14, Stratford place, Oxford street.
- 1889 JOHNSON, RAYMOND, M.B., B.S., Assistant Surgeon to University College Hospital; Surgeon to the Victoria Hospital for Children; 20, Weymouth street. *Trans.* 1.
- 1884 JOHNSTON, JAMES, M.D., 11, Chester place, Hyde Park square.

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- 1887 JONES, HENRY LEWIS, M.D., Medical Officer in charge of Electrical Department at St. Bartholomew's Hospital; 9, Upper Wimpole street, Cavendish square.
- 1881 JULER, HENRY EDWARD, Ophthalmic Surgeon to St. Mary's Hospital; Surgeon to the Royal Westminster Ophthalmic Hospital; Consulting Ophthalmic Surgeon to the London Lock Hospital; 23, Cavendish square.
- 1893 KANTHACK, ALFRED A., M.D., Lecturer on Pathology, St. Bartholomew's Hospital.
- 1882 KEETLEY, CHARLES R. B., Senior Surgeon to the West London Hospital; 56, Grosvenor street, Grosvenor square.
- 1884 KESER, JEAN SAMUEL, M.D., Physician to the French Hospital; 11, Harley street, Cavendish square.
- †1857 KIALLMARK, HENRY WALTER, 5, Pembridge gardens. C. 1890-91.
- 1881 KIDD, PERCY, M.A., M.D., Physician to the Hospital for Consumption, Brompton; Assistant Physician to the London Hospital; 60, Brook street, Grosvenor square. *Trans.* 4.
- †1851 KINGDON, JOHN ABERNETHY, Consulting Surgeon to the Bank of England; 2, Bank buildings, Lothbury. C. 1866-7. V.P. 1872-3. *Sci. Com.* 1867. *Trans.* 1.
- 1884 LANE, WILLIAM ARBUTHNOT, M.S., Lecturer on Anatomy at Guy's Hospital; Assistant Surgeon to the Hospital for Sick Children; 8, St. Thomas's street, Southwark. *Trans.* 4.
- 1882 LANG, WILLIAM, Ophthalmic Surgeon to, and Lecturer on Ophthalmic Surgery at, the Middlesex Hospital; Surgeon to the Royal London Ophthalmic Hospital, Moorfields; 22, Cavendish square.
- †1863 LANGDON-DOWN, JOHN LANGDON HAYDON, M.D., Consulting Physician to the London Hospital; 81, Harley street, Cavendish square. C. 1880. V.P. 1890-91. *Trans.* 2.

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- 1894 LANGDON-DOWN, REGINALD LANGDON, M.B., B.C., 81, Harley street.
- †1865 LANGTON, JOHN, Surgeon to, and Lecturer on Clinical Surgery at, St. Bartholomew's Hospital; Surgeon to the City of London Truss Society; 62, Harley street, Cavendish square. C. 1881-2. V.P. 1895—. *Referee*, 1885-95. *Lib. Com.* 1879-80, 1888—. *Trans.* 2.
- 1890 LAW, EDWARD, M.D., C.M., 35, Harley street, Cavendish square.
- 1888 LAWRENCE, LAURIE ASHER, 125, Harley street, Cavendish square.
- 1893 LAWSON, ARNOLD, 12, Harley street.
- 1884 LAWSON, GEORGE, Surgeon-Oculist in Ordinary to H.M. the Queen; Consulting Surgeon to the Royal London Ophthalmic Hospital; Surgeon to the Middlesex Hospital; 12, Harley street, Cavendish square.
- 1892 LEADAM, WILLIAM WARD, M.D., 80, Gloucester terrace, Hyde Park.
- †1843 LEE, HENRY, Consulting Surgeon to St. George's Hospital; 9, Savile row, Burlington gardens. C. 1856-7. L. 1863-4. V.P. 1868-9. *Referee*, 1855, 1866-8. *Sci. Com.* 1867. *Trans.* 14. *Pro.* 2.
- 1895 LESLIE, ROBERT MURRAY, M.B., 58, Harley street.
- 1886 LEWERS, ARTHUR HAMILTON NICHOLSON, M.D., Obstetric Physician to the London Hospital; 60, Wimpole street, Cavendish square. *Trans.* 1.
- 1878 LISTER, SIR JOSEPH, Bart., D.C.L., LL.D., F.R.S., Surgeon Extraordinary to H.M. the Queen; Emeritus Professor of Clinical Surgery in King's College, London; and Consulting Surgeon to King's College Hospital; 12, Park crescent, Regent's Park. C. 1892.
- 1891 LITTLE, ERNEST MUIRHEAD, 40, Seymour street, Portman square.
- 1889 LITTLE, JOHN FLETCHER, M.B., 32, Harley street, Cavendish square.

*Elected*

- 1881 LOCKWOOD, CHARLES BARRETT, Surgeon to the Great Northern Central Hospital; Assistant Surgeon to, and Demonstrator of Operative Surgery at, St. Bartholomew's Hospital; 19, Upper Berkeley street, Portman square. *Trans.* 4.
- 1881 LUCAS, RICHARD CLEMENT, B.S., M.B., Surgeon to, and Lecturer on Anatomy at, Guy's Hospital; Surgeon to the Evelina Hospital for Sick Children; Corresponding Member of the Société de Chirurgie of Paris; 50, Wimpole street, Cavendish square. *Trans.* 2.
- 1888 LUFF, ARTHUR PEARSON, M.D., B.Sc., Physician to Out-patients and Lecturer on Medical Jurisprudence at St. Mary's Hospital; Official Analyst to the Home Office; 31, Weymouth street, Portland place.
- 1887 LUSH, PERCY J. F., M.B., 4, Maresfield gardens, Hampstead.
- †1873 MACCARTHY, JEREMIAH, M.A., Surgeon to the London Hospital, late Lecturer on Surgery at the London Hospital Medical College; 15, Finsbury square. C. 1886-7. *Lib. Com.* 1882-5. *Referee*, 1890—.
- 1867 MAC CORMAC, SIR WILLIAM, M.A., M.Ch., D.Sc., Consulting Surgeon to St. Thomas's Hospital; 13, Harley street. C. 1884-5. *Referee*, 1889—. *Lib. Com.* 1895—. *Trans.* 1.
- 1894 MACFADYEN, ALLAN, M.D., B.S., 101, Great Russell street.
- 1880 MCHARDY, MALCOLM MACDONALD, Ophthalmic Surgeon to King's College Hospital, and Professor of Ophthalmic Surgery in King's College, London; Surgeon to the Royal Eye Hospital, Southwark; 5, Savile row.
- 1873 MACKELLAR, ALEXANDER OBERLIN, M.Ch., Surgeon to St. Thomas's Hospital; Surgeon-in-Chief to the Metropolitan Police Force; 79, Wimpole street, Cavendish square.

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- 1881 **MACKENZIE, STEPHEN, M.D.**, Physician to the London Hospital, and Lecturer on the Principles and Practice of Medicine at the London Hospital Medical College; Physician to the Royal London Ophthalmic Hospital; 18, Cavendish square. *Referee*, 1890—. *Trans.* 1.
- 1879 **MACLAGAN, THOMAS JOHN, M.D.**, Physician-in-Ordinary to their R.H. the Prince and Princess Christian of Schleswig-Holstein; 9, Cadogan place, Belgrave square.
- 1889 **MACLEHOSE, NORMAN MACMILLAN, M.B., C.M.**, 13, Queen Anne street, Cavendish square.
- 1893 **MCLEOD, KENNETH, M.D.**, 39, Clanricarde gardens, Bayswater. *Trans.* 1.
- 1876 **MACNAMARA, N. CHARLES**, Surgeon to, and Lecturer on Surgery at, the Westminster Hospital; Consulting Surgeon to the Royal Westminster Ophthalmic Hospital; 13, Grosvenor street. C. 1891-2. *Referee*, 1884-90, 1895—. *Lib. Com.* 1886-90.
- 1881 **MACBEADY, JONATHAN FORSTER CHRISTIAN HORACE**, Surgeon to the Great Northern Hospital; 51, Queen Anne street, Cavendish square.
- 1880 **MADDICK, EDMUND DISTIN**, 2, Chandos street, Cavendish square.
- 1886 **MAGUIRE, ROBERT, M.D.**, Physician to Out-patients and Joint Lecturer on Pathology at St. Mary's Hospital; Assistant Physician to the Hospital for Consumption, Brompton; 4, Seymour street, Portman square. *Sci. Com.* 1889—.
- 1880 **MAKINS, GEORGE HENRY**, Assistant Surgeon to St. Thomas's Hospital; Surgeon to the Evelina Hospital for Children; 47, Charles street, Berkeley square. *Trans.* 1.
- 1885 **MALCOLM, JOHN DAVID, M.B., C.M.**, Surgeon to the Samaritan Free Hospital; 13, Portman street, Portman square. *Trans.* 2.



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- 1890 MANSON, PATRICK, M.D., C.M., LL.D., Physician to the Seamen's Hospital, Greenwich ; 21, Queen Anne street, Cavendish square.
- 1855 MARCET, WILLIAM, M.D., F.R.S., Flowermead, Wimbledon Park. C. 1871. *Referee*, 1866-70, 1883-6. *Sci. Com.* 1863. *Lib. Com.* 1866-8. *Trans.* 3.
- 1867 MARSH, F. HOWARD, Surgeon to, and Lecturer on Surgery at, St. Bartholomew's Hospital; 30, Bruton street, Berkeley square. C. 1882-3, 1889. S. 1885-7. V.P. 1891-3. *Lib. Com.* 1880-1. *Trans.* 4.
- 1891 MARTIN, HENRY CHARRINGTON, M.D., 27, Oxford square.
- 1884 MARTIN, SIDNEY HARRIS COX, M.D., F.R.S., Assistant Physician to University College Hospital, and to the Hospital for Consumption, Brompton ; 10, Mansfield street, Portland place.
- 1892 MASTERS, JOHN ALFRED, M.D., 57, Lexham gardens, Kensington.
- 1891 MAY, WILLIAM PAGE, M.D., B.Sc., 38, Weymouth street.
- 1891 MERCIER, CHARLES ARTHUR, M.B., Lecturer on Neurology and Insanity to Westminster Hospital ; 8, New Court, Lincoln's Inn, and Flower House, Southend, Catford.
- 1880 MEREDITH, WILLIAM APPLETON, M.B., C.M., Surgeon to the Samaritan Free Hospital for Women and Children ; 21, Manchester square. *Trans.* 1.
- 1894 MICHELS, ERNST, M.D., 6, West street, Finsbury circus. *Trans.* 1.
- 1893 MILEY, MILES, M.B., 21, Belsize avenue, Hampstead.
- 1891 MOLINE, PAUL, M.B., 42, Walton street, Chelsea.
- 1873 MOORE, NORMAN, M.D., Assistant Physician to, and Joint Lecturer on Medicine at, St. Bartholomew's Hospital; 94, Gloucester place, Portman square. C. 1891-2. *Referee*, 1886-90. *Sci. Com.* 1889—.

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- 1878 MORGAN, JOHN HAMMOND, M.A., Surgeon to the Charing Cross Hospital, and to the Hospital for Sick Children, Great Ormond street ; 68, Grosvenor street. C. 1895—. *Trans.* 2.
- 1894 MORISON, ALEXANDER, M.D., 14, Upper Berkeley street, W.
- 1874 MORRIS, HENRY, M.A., Surgeon to, and Lecturer on Surgery at, the Middlesex Hospital ; 8, Cavendish square. C. 1888-9. *Referee*, 1882-7. *Lib. Com.* 1895—. *Trans.* 10.
- 1879 MORRIS, MALCOLM ALEXANDER, Surgeon to the Skin Department of, and Lecturer on Dermatology at, St. Mary's Hospital ; 8, Harley street, Cavendish square. *Sci. Com.* 1889—. *Trans.* 1.
- 1885 MOTT, FREDERICK WALKER, M.D., Assistant Physician to Charing Cross Hospital ; Pathologist to the London County Council ; 84, Wimpole street, Cavendish square.
- †1888 MURRAY, HUBERT MONTAGUE, M.D., Physician to Out-patients, and Lecturer on Pathology at, the Charing Cross Hospital ; 27, Savile row.
- 1880 MURRELL, WILLIAM, M.D., Physician to Out-patients, and Lecturer on Materia Medica and Therapeutics, at the Westminster Hospital ; 17, Welbeck street, Cavendish square. *Sci. Com.* 1889—. *Trans.* 1.
- 1892 MYDDELTON-GAVEY, E. HERBERT, 94, Wimpole street, Cavendish square.
- 1863 MYERS, ARTHUR BOWEN RICHARDS, late Brigade-Surgeon, Brigade of Guards ; 43, Gloucester street, Warwick square. C. 1878-9. *Lib. Com.* 1877.
- 1877 NETTLESHIP, EDWARD, Ophthalmic Surgeon to, and Lecturer on Ophthalmology at, St. Thomas's Hospital ; Surgeon to the Royal London Ophthalmic Hospital ; 5, Wimpole street, Cavendish square. *Referee*, 1892—.
- 1864 NUNN, THOMAS WILLIAM, Consulting Surgeon to the Middlesex Hospital ; 8, Stratford place, Oxford street.
- 1880 OGILVIE, GEORGE, M.B., B.Sc., Physician to the Hospital for Epilepsy and Paralysis, Regent's Park ; 22, Welbeck street, Cavendish square.

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- 1880 OGILVIE, LESLIE, M.B., B.Sc., Physician to the Paddington Green Children's Hospital; 46, Welbeck street, Cavendish square.
- 1891 OGLE, CYRIL, M.A., M.B., 30, Cavendish square.
- 1858 OGLE, JOHN WILLIAM, M.A., M.D., Consulting Physician to St. George's Hospital; 30, Cavendish square. C. 1873. V.P. 1886. *Referee*, 1864-72. *Trans.* 4.
- 1860 OGLE, WILLIAM, M.D., late Superintendent of Statistics in the Registrar-General's Department, Somerset House; 10, Gordon street, Gordon square. S. 1868-70. C. 1876-7. V.P. 1887. *Lib. Com.* 1871-5. *Trans.* 5.
- 1892 OPENSHAW, T. HORROCKS, M.B., M.S., Assistant Surgeon to, and Lecturer on Anatomy at, the London Hospital; 16, Wimpole street, Cavendish square.
- 1873 ORD, WILLIAM MILLER, M.D., Physician to, and Lecturer on Medicine at, St. Thomas's Hospital; 37, Upper Brook street, Grosvenor square. C. 1889-90. *Sci. Com.* 1889—. *Referee*, 1884-8. *Trans.* 6.
- 1890 ORD, WILLIAM WALLIS, M.D., 2, Queen street, Mayfair.
- 1877 ORMEROD, JOSEPH ARDERNE, M.D., Assistant Physician to St. Bartholomew's Hospital; Physician to the National Hospital for the Paralysed and Epileptic, Queen square; 25, Upper Wimpole street. *Trans.* 1.
- 1875 OSBORN, SAMUEL C., 10, Maddox street, Regent street, and Maisonnnette, Datchet, Berks.
- 1879 OWEN, EDMUND, M.B., Surgeon to, and Joint Lecturer on Surgery at, St. Mary's Hospital; Senior Surgeon to the Hospital for Sick Children, Great Ormond street; 64, Great Cumberland place, Hyde park. *Trans.* 3.
- 1882 OWEN, HERBERT ISAMBAARD, M.D., Physician to, and Lecturer on Forensic Medicine at, St. George's Hospital; 40, Curzon street, Mayfair. *Bldg. Com.* 1889-92. *Referee*, 1893, 1895—.

*Elected*

- 1892 PAGE, HARRY MARMA DUKE, 82, Ashley gardens, Victoria street.
- 1874 PAGE, HERBERT WILLIAM, M.A., M.C., Surgeon to, and Joint Lecturer on Surgery at, St. Mary's Hospital; 146, Harley street, Cavendish square. C. 1890-91. *Referee*, 1884-89. *Lib. Com.* 1886-8. *Trans.* 4.
- †1840 PAGET, SIR JAMES, Bart., D.C.L., LL.D., F.R.S., Sergeant-Surgeon to H.M. the Queen; Surgeon-in-Ordinary to H.R.H. the Prince of Wales; Consulting Surgeon to St. Bartholomew's Hospital; Vice-Chancellor of the University of London; Foreign Associate of the Académie de Médecine, Paris; 5, Park square West, Regent's park. C. 1848-9. V.P. 1861. T. 1867. P. 1875-6. *Referee*, 1844-6, 1848, 1851-60, 1862-6, 1868-74. *Sci. Com.* 1863. *Lib. Com.* 1846-7. *Trans.* 12.
- 1886 PAGET, STEPHEN, Surgeon to, and Surgeon to the Aural Department at, the West London Hospital; 57, Wimpole street, Cavendish square.
- 1895 PARKER, CHARLES ARTHUR, 41, Queen Anne street, Cavendish square.
- 1873 PARKER, ROBERT WILLIAM, *Hon. Secretary*; Senior Surgeon to the East London Hospital for Children; Senior Surgeon to the German Hospital; 13, Welbeck street, Cavendish square. C. 1888-9. S. 1895—. *Bldg. Com.* 1889-92. *Referee*, 1891-5. *Lib. Com.* 1885-87, 1892-5. *Ho. Com.* 1892-5. *Trans.* 4.
- 1889 PARSONS, J. INGLIS, M.D., Physician to Out-patients, Chelsea Hospital for Women; 3, Queen street, Mayfair.
- 1883 PASTEUR, WILLIAM, M.D., Assistant Physician to the Middlesex Hospital; Physician to the North-Eastern Hospital for Children; 4, Chandos street, Cavendish square.
- 1891 PATERSON, WILLIAM BROMFIELD, 64, Brook street, Grosvenor square.
- 1891 PATON, EDWARD PERCY, M.D., St. Bartholomew's Hospital.

*Elected*

- 1865 PAVY, FREDERICK WILLIAM, M.D., LL.D., F.R.S., Consulting Physician to Guy's Hospital; 35, Grosvenor street. C. 1883-4. V.P. 1893-4. *Referee*, 1871-82. *Trans.* 1.
- 1869 PAYNE, JOSEPH FRANK, M.D., Physician to, and Lecturer on Medicine at, St. Thomas's Hospital; 78, Wimpole street, Cavendish square. C. 1887. *Referee*, 1890—. *Sci. Com.* 1879. *Lib. Com.* 1878-85, 1889—.
- 1894 PEGLER, L. HEMINGTON, M.D., 12, Radnor place, Gloucester square.
- 1887 PENROSE, FRANCIS GEORGE, M.D., Assistant Physician to St. George's Hospital; 4, Harley street, Cavendish square. *Sci. Com.* 1889—.
- 1890 PERRY, EDWIN COOPER, M.D., Assistant Physician to, and Demonstrator of Pathology at, Guy's Hospital; The College, Guy's Hospital.
- 1883 PHILLIPS, CHARLES DOUGLAS F., M.D., LL.D., 10, Henrietta street, Cavendish square.
- 1884 PHILLIPS, GEORGE RICHARD TURNER, 24, Palace Court, Notting hill gate.
- 1888 PHILLIPS, JOHN, M.A., M.D., Assistant Obstetric Physician, King's College Hospital; Physician to the British Lying-in Hospital; 71, Grosvenor street, Grosvenor square. *Trans.* 1.
- 1889 PHILLIPS, SIDNEY, M.D., Senior Physician to Out-patients and Lecturer on Materia Medica at St. Mary's Hospital; Senior Physician to the London Fever Hospital, and to the Lock Hospital; 62, Upper Berkeley street, Portman square.
- 1867 PICK, THOMAS PICKERING, Surgeon to, and Lecturer on Surgery at, St. George's Hospital; 18, Portman street, Portman square. C. 1884-5. V.P. 1893-4. *Referee*, 1882-3, 1891-93. *Sci. Com.* 1870, 1889—. *Lib. Com.* 1879-81.
- 1884 PITT, GEORGE NEWTON, M.D., Assistant Physician to, and Pathologist at, Guy's Hospital; 24, St. Thomas's street, Southwark. *Trans.* 1.

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- 1889 PITTS, BERNARD, M.B., M.C., Surgeon to St. Thomas's Hospital; 109, Harley street, Cavendish square.
- 1885 POLAND, JOHN, Surgeon to the Miller Hospital, Greenwich; 4, St. Thomas's street, Southwark.
- 1884 POLLARD, BILTON, B.S., Surgeon to University College Hospital, Surgeon to the North-Eastern Hospital for Children; 24, Harley street, Cavendish square. *Trans.* 1.
- †1845 POLLOCK, GEORGE DAVID, Surgeon-in-Ordinary to H.B.H. the Prince of Wales; Consulting Surgeon to St. George's Hospital; 35, Chester square. C. 1856-7. L. 1859-62. V.P. 1870-1. P. 1886-7. *Referee*, 1858, 1864-9, 1877-85. *Trans.* 5.
- 1865 POLLOCK, JAMES EDWARD, M.D., Consulting Physician to the Hospital for Consumption, Brompton; 52, Upper Brook street, Grosvenor square. C. 1882-3. *Referee*, 1872-81.
- 1894 POLLOCK, WILLIAM RIVERS, M.B., B.C., 56, Park street, Grosvenor square.
- 1871 POORE, GEORGE VIVIAN, M.D., Professor of Medical Jurisprudence in University College, London; Physician to University College Hospital; Consulting Physician to the Royal Infirmary for Children and Women, Waterloo road; 30, Wimpole street, Cavendish square. C. 1890-91. *Referee* 1887-89, 1892—. *Lib. Com.* 1895—. *Trans.* 2.
- 1885 PORT, HEINRICH, M.D., Physician to the German Hospital; 48, Finsbury square.
- 1867 POWELL, RICHARD DOUGLAS, M.D., Physician Extraordinary to H.M. the Queen; Physician to the Middlesex Hospital; Consulting Physician to the Hospital for Consumption, Brompton; 62, Wimpole street, Cavendish square. S. (Oct.) 1883-5. C. 1887-8. *Referee*, 1879-83, 1886. *Trans.* 3.

*Elected*

- 1887 POWER, D'ARCY, M.A., M.B., Demonstrator of Surgery at St. Bartholomew's Hospital; Surgeon to the Victoria Hospital for Children; 26, Bloomsbury square. *Trans.* 2.
- 1867 POWER, HENRY, Consulting Ophthalmic Surgeon to St. Bartholomew's Hospital; 37A, Great Cumberland place, Hyde Park. C. 1882-3. V.P. 1892-3. *Referee*, 1870-81, 1891—. *Sci. Com.* 1870, 1889—. *Lib. Com.* 1872-8.
- †1857 PRIESTLEY, SIR WILLIAM OVEREND, M.D., LL.D., Consulting Physician to King's College Hospital, and to the West London Hospital and the British Lying-in Hospital; 17, Hertford street, Mayfair. C. 1874-5. V.P. 1884-5. *Referee*, 1867-'3, 1877-83. *Sci. Com.* 1863.
- 1883 PRINGLE, JOHN JAMES, M.B., C.M., Lecturer on Practical Medicine, Physician in Charge of Skin Department at, the Middlesex Hospital; 23, Lower Seymour street, Portman square. *Trans.* 2.
- 1874 PURVES, WILLIAM LAIDLAW, Aural Surgeon to Guy's Hospital; 20, Stratford place, Oxford street. *Trans.* 2.
- 1877 PYE-SMITH, PHILIP HENRY, M.D., F.R.S., Physician to, and Lecturer on Medicine at, Guy's Hospital; Member of the Senate of the University of London; 48, Brook street, Grosvenor square. C. 1893-94. *Lib. Com.* 1887-93. *Trans.* 1.
- †1850 QUAIN, SIR RICHARD, Bart., M.D., (Hon.) M.D. Dublin, LL.D. Ed., F.R.S., Physician Extraordinary to H.M. the Queen; President of the General Medical Council; Consulting Physician to the Hospital for Consumption, Brompton; Member of the Senate of the University of London; 67, Harley street, Cavendish square. C. 1866-7. V.P. 1878-9. *Sci. Com.* 1863. *Trans.* 1.

*Elected*

- 1871 RALFE, CHARLES HENRY, M.D., M.A., Physician to the London Hospital, and late Physician to the Seamen's Hospital, Greenwich ; 26, Queen Anne street, Cavendish square. C. 1889. *Referee*, 1885-8.
- 1892 RAYNER, HENRY, M.D., 2, Harley street, Cavendish square.
- 1869 READ, THOMAS LAURENCE, 11, Petersham terrace, Queen's gate.
- 1891 REECE, RICHARD JAMES, 31, Holland Villas road, W.
- 1891 REMFRY, LEONARD, M.A., M.D., Obstetrical Physician to the Great Northern Central Hospital; Assistant Obstetric Physician to, and Assistant Lecturer on Obstetric Medicine at, St. George's Hospital; 60, Great Cumberland place.
- 1891 RENDEL, ARTHUR BOWEN, M.A., M.B., B.C., 43, Albion street, Hyde Park, W.
- †1855 REYNOLDS, SIR JOHN RUSSELL, Bart., M.D., F.R.S., Physician-in-Ordinary to H.M.'s Household; Emeritus Professor of Medicine in University College; Consulting Physician to University College Hospital; 38, Grosvenor street. C. 1870. V.P. 1883. *Referee*, 1867-9.
- 1887 RICHARDSON, GILBERT, M.A., M.D., Hawthorn House, Putney.
- 1863 RINGER, SYDNEY, M.D., F.R.S., Holme Professor of Clinical Medicine in University College, London, and Physician to University College Hospital; 15, Cavendish place, Cavendish square. C. 1881-2. *Referee*, 1873-80, 1889—. *Trans.* 6.
- 1871 RIVINGTON, WALTER, M.S., Consulting Surgeon to the London Hospital; 95, Wimpole street, Cavendish square. C. 1885-6. *Trans.* 5.
- 1893 ROBERTS, D. WATKIN, M.D., 56, Manchester street, Manchester square.
- 1878 ROBERTS, FREDERICK THOMAS, M.D., Professor of Materia Medica and Therapeutics, and of Clinical Medicine, in University College, London; Physician to University College Hospital; Consulting Physician to the Hospital for Consumption, Brompton; 102, Harley street, Cavendish square. C. 1894-5. *Sci. Com.* 1889—.



*Elected*

- 1889 ROBERTS, SIR WILLIAM, M.D., B.A., F.R.S., 8, Manchester square. *Trans.* 2.
- 1890 ROLLESTON, HUMPHRY DAVY, M.A., M.D., Assistant Physician to, and Lecturer on Pathology at, St. George's Hospital; 13, Upper Wimpole street, Cavendish square.
- †1857 ROSE, HENRY COOPER, M.D., Consulting Surgeon to the Hampstead Dispensary; 53, Roslyn hill, Hampstead. C. 1886-7. *Trans.* 1.
- 1883 ROSE, WILLIAM, M.B., Professor of Clinical Surgery in King's College; Surgeon to King's College Hospital; and Consulting Surgeon to the Royal Free Hospital; 17, Harley street, Cavendish square.
- 1888 ROUGHTON, EDMUND WILKINSON, B.S., M.D., Warden of the College of St. Mary's Hospital; Assistant Surgeon to the Royal Free Hospital; 33, Westbourne terrace, Hyde Park. *Trans.* 1.
- 1882 ROUTH, AMAND JULES McCONNEL, M.D., B.S., Physician to the Samaritan Free Hospital for Women and Children; Obstetric Physician to Out-patients, and Lecturer on Practical Midwifery, at the Charing Cross Hospital; 14A, Manchester square.
- †1849 ROUTH, CHARLES HENRY FELIX, M.D., Consulting Physician to the Samaritan Free Hospital for Women and Children; 52, Montagu square. *Lib. Com.* 1854-5. *Trans.* 1.
- 1891 RUFFER, MARC ARMAND, M.A., M.D., 5, York terrace, Regent's park.
- 1891 RUSSELL, J. S. RISIEN, M.B., C.M., 4, Queen Anne street, Cavendish square.
- 1886 SAINSBURY, HARRINGTON, M.D., Physician to the Royal Free Hospital; Assistant Physician to the City of London Hospital for Diseases of the Chest; 63, Welbeck street, Cavendish square. *Trans.* 1.
- †1869 SANSOM, ARTHUR ERNEST, M.D., Physician to the London Hospital; Consulting Physician, North - Eastern Hospital for Children; 84, Harley street, Cavendish square. C. 1887-8. *Referee*, 1889—. *Trans.* 3.

*Elected*

- †1845 SAUNDERS, SIR EDWIN, Surgeon-Dentist to H.M. the Queen, and to their R.H. the Prince and Princess of Wales; Fairlawn, Wimbledon Common. C. 1872-3.
- 1879 SAVAGE, GEORGE HENRY, M.D., Lecturer on Mental Diseases at Guy's Hospital; 3, Henrietta street, Cavendish square.
- 1883 SCHÄFER, EDWARD ALBERT, F.R.S., Jodrell Professor of Physiology, University College, London; University College, Gower street. *Referee*, 1888—. *Sci. Com.* 1889—.
- 1892 SCHORSTEIN, GUSTAVE, M.A., M.B., B.Ch., D.P.H., Assistant Physician to the London Hospital, and to the Hospital for Consumption, Brompton; 11, Portland place.
- 1887 SCOTT, HARRY, M.D., 27, St. Ermin's mansions, Westminster.
- 1882 SCRIVEN, JOHN BARCLAY, Brigade Surgeon, Bengal (retired), late Professor of Anatomy, Surgery, and Ophthalmic Surgery at the Lahore Medical School; 95, Oxford gardens, Notting hill.
- 1863 SEDGWICK, WILLIAM, 101, Gloucester place, Portman square. C. 1884-5. *Trans.* 3.
- 1892 DE SEGUNDO, CHARLES SEMPILL, 2, Aldridge road villas, Westbourne park.
- 1892 SELWYN-HARVEY, JOHN STEPHENSON, M.D., 1, Astwood road, Cromwell road.
- 1877 SEMON, FELIX, M.D., Physician for Diseases of the Throat to St. Thomas's Hospital; 39, Wimpole street, Cavendish square. C. 1895—. *Lib. Com.* 1894—. *Trans.* 3.
- 1894 SEWILL, JOSEPH SEFTON, 9A, Cavendish square.
- 1882 SHARKEY, SEYMOUR JOHN, M.D., Physician to, and Joint Lecturer on Medicine at, St. Thomas's Hospital; 2, Portland place. *Trans.* 2.
- 1886 SHAW, LAURISTON ELGIE, M.D., Assistant Physician to Guy's Hospital; 10, St. Thomas's street, Southwark.
- 1884 SHEILD, ARTHUR MARMADUKE, M.B., B.C., Assistant Surgeon to St. George's Hospital; 4, Cavendish place. *Trans.* 3.

*Elected*

- 1893 SIBLEY, WALTER KNOWSLEY, M.D., 7, Upper Brook street.
- †1848 SIEVEKING, SIR EDWARD HENRY, M.D., LL.D., F.S.A., Physician-in-Ordinary to H.M. the Queen; Physician-in-Ordinary to H.R.H. the Prince of Wales; Consulting Physician to St. Mary's and the Lock Hospitals; 17, Manchester square. C. 1859-60. S. 1861-3. V.P. 1873-4. L. 1881-2. P. 1888-9. *Referee*, 1855-8, 1864-72, 1875-80. *Sci. Com.* 1862. *Trans.* 2.
- 1886 SILCOCK, ARTHUR QUARRY, B.S., Surgeon in charge of Out-patients, St. Mary's Hospital; Surgeon to the Royal London Ophthalmic Hospital; 52, Harley street, Cavendish square. *Lib. Com.* 1895—.
- †1842 SIMON, SIR JOHN, K.C.B., D.C.L., LL.D., F.R.S., Consulting Surgeon to St. Thomas's Hospital; 40, Kensington square. C. 1854-5. V.P. 1865. *Referee*, 1851-3, 1866-81. *Trans.* 1.
- 1892 SIMS, FRANCIS MANLEY BOLDERO, 12, Hertford street, Mayfair.
- 1893 SISLEY, RICHARD, M.D., 11, York street, Portman square.
- 1894 SLATER, CHARLES, M.B., 16, Northwick terrace, St. John's Wood.
- 1890 SMALE, MORTON, 22A, Cavendish square.
- 1879 SMITH, E. NOBLE, Surgeon to All Saints' Children's Hospital; Orthopædic Surgeon to the British Home for Incurables; 24, Queen Anne street, Cavendish square.
- 1881 SMITH, EUSTACE, M.D., Physician to H.M. the King of the Belgians; Physician to the East London Children's Hospital, and to the Victoria Park Hospital for Diseases of the Chest; 15, Queen Anne street, Cavendish square.
- 1891 SMITH, G. COCKBURN, M.D., 5, Inverness gardens, Kensington.

*Elected*

- †1838 SMITH, HENRY SPENCER, Consulting Surgeon to St. Mary's Hospital; 92, Oxford terrace, Hyde Park. C. 1854. S. 1855-8. V.P. 1859-60. T. 1865. *Referee*, 1851-3, 1862-4, 1866-78. *Lib. Com.* 1847.
- 1866 SMITH, HEYWOOD, M.D., 18, Harley street, Cavendish square.
- 1889 SMITH, ROBERT PERCY, M.D., B.S., Resident Physician and Medical Superintendent, Bethlem Royal Hospital, St. George's road, Southwark.
- 1892 SMITH, SOLOMON CHARLES, M.D., 4, Portman Mansions Baker street.
- 1863 SMITH, THOMAS, Surgeon to, and Lecturer on Clinical Surgery at, St. Bartholomew's Hospital; 5, Stratford place, Oxford street. S. 1870-2. C. 1875-6. V.P. 1887-8. *Referee*, 1873-4, 1880-6. *Sci. Com.* 1867. *Trans.* 4.
- 1872 SMITH, THOMAS GILBERT, M.D., Physician to the London Hospital; Physician to the Royal Hospital for Diseases of the Chest, City road; 68, Harley street, Cavendish square. C. 1890. *Trans.* 1.
- 1894 SMITH, THOMAS RUDOLPH, M.B., B.C., 5, Stratford place, Oxford street.
- 1873 SMITH, W. JOHNSON, Surgeon to the Seamen's Hospital Society, Greenwich.
- \*1874 SMITH, WILLIAM ROBERT, M.D., D.Sc., Barrister-at-Law, Professor of Forensic Medicine in, and Director of the Laboratories of State Medicine at, King's College, London; Medical Officer to the School Board for London; 74, Great Russell Street. *Trans.* 1.
- 1865 SOUTHEY, REGINALD, M.D., Commissioner in Lunacy; 32, Grosvenor road, Westminster. C. 1881-2. S. 1883. *Referee*, 1873-80. *Trans.* 1.
- 1889 SPENCER, HERBERT R., M.D., B.S., Professor of Midwifery in University College; Obstetric Physician to University College Hospital; 10, Mansfield street, Portland place. *Referee*, 1894—.

*Elected*

- 1887 SPENCER, WALTER GEORGE, M.B., M.S., Assistant Surgeon to the Westminster Hospital; 35, Brook street, Grosvenor square. *Trans.* 2.
- 1888 SPICER, ROBERT HENRY SCANES, M.D., Physician to the Department for Diseases of the Throat, St. Mary's Hospital; 28, Welbeck street, Cavendish square.
- 1890 SPICER, WILLIAM THOMAS HOLMES, M.B., 47, Welbeck street, Cavendish square.
- 1875 SPITTA, EDMUND JOHNSON, Ivy House, Clapham Common, Surrey.
- †1851 SPITTA, ROBERT JOHN, M.D., 2, The Sweep, East Side, Clapham Common, Surrey. C. 1878-9. *Trans.* 1.
- 1885 SQUIRE, JOHN EDWARD, M.D., Physician to the North London Hospital for Consumption; 122, Harley street, Cavendish square. *Trans.* 2.
- †1859 STEWART, WILLIAM EDWARD, 16, Harley street, Cavendish square.
- †1856 STOCKER, ALONZO HENRY, M.D., Peckham House, Peckham.
- 1884 STONHAM, CHARLES, Assistant Surgeon to the Westminster Hospital, and Curator of Anatomical Museum; 4, Harley street, Cavendish square.
- †1871 SUTHERLAND, HENRY, M.D., Physician to Newland's House and Otto House Private Asylums; 6, Richmond terrace, Whitehall.
- 1883 SUTTON, JOHN BLAND, Assistant Surgeon to the Middlesex Hospital; 48, Queen Anne street, Cavendish square. *Trans.* 6.
- 1890 SYERS, HENRY WALTER, M.D., 3, Devonshire street, Portland place.
- 1886 SYMONDS, CHARTERS JAMES, M.S., Assistant Surgeon to, and Demonstrator of Operative and Practical Surgery at, Guy's Hospital; 26, Weymouth street, Portland place.

*Elected*

- 1875 TAY, WARREN, Senior Surgeon to the London Hospital ; Surgeon to the Royal London Ophthalmic Hospital ; Consulting Surgeon to the North-Eastern Hospital for Children, and to the Hospital for Diseases of the Skin, Blackfriars ; 4, Finsbury square.
- 1873 TAYLOR, FREDERICK, M.D., *Trustee* ; Physician to, and Lecturer on Medicine at, Guy's Hospital ; Consulting Physician to the Evelina Hospital for Sick Children ; 20, Wimpole street, Cavendish square. S. 1889-93. C. 1894-5. *Sci. Com.* 1889—. *Referee*, 1887-8. *Trans.* 3.
- 1893 TAYLOR, JAMES, M.D., Assistant Physician to the National Hospital for the Paralysed and Epileptic ; 34, Welbeck street, Cavendish square. *Trans.* 1.
- 1890 TAYLOR, SEYMOUR, M.D., Assistant Physician West London Hospital ; 16, Seymour street, Portman square.
- 1859 TEGART, EDWARD, 60, Scarsdale Villas, Kensington. C. 1888-9.
- 1874 THIN, GEORGE, M.D., 22, Queen Anne street, Cavendish square. C. 1893-4. *Trans.* 11.
- †1862 THOMPSON, EDMUND SYMES, M.D., Consulting Physician to the Hospital for Consumption, Brompton ; Gresham Professor of Medicine ; 33, Cavendish square. S. 1871-4. C. 1878-9. *Sci. Com.* 1889—. *Referee*, 1876-7. *Trans.* 1.
- †1852 THOMPSON, SIR HENRY, Surgeon-Extraordinary to H.M. the King of the Belgians ; Emeritus Professor of Clinical Surgery in University College, London ; and Consulting Surgeon to University College Hospital ; Member of the Société de Chirurgie, Paris ; 35, Wimpole street, Cavendish square. C. 1869. *Trans.* 8.
- †1862 THOMPSON, REGINALD EDWARD, M.D., Consulting Physician to the Hospital for Consumption, Brompton ; 47, Park street, Grosvenor square. C. 1879. S. 1880-82. V.P. 1893-4. *Referee*, 1873-8. *Sci. Com.* 1867. *Trans.* 2.

*Elected*

- 1892 THOMSON, ST. CLAIR, M.D., 28, Queen Anne street, Cavendish square. *Trans.* 1.
- 1892 THORNE, WILLIAM BEZLY, M.D., 53, Upper Brook Street.
- 1876 THORNTON, JOHN KNOWSLEY, M.B., C.M., Consulting Surgeon to the Samaritan Free Hospital for Women and Children; Consulting Surgeon to the Grosvenor Hospital for Women, and to the New Hospital for Women; 49, Montagu square. C. 1891. *Lib. Com.* 1886-90, 1893—. *Trans.* 5.
- 1889 TIRARD, NESTOR ISIDORE CHARLES, M.D., Professor of Materia Medica and Therapeutics, King's College; Physician to King's College Hospital, and Physician to the Evelina Hospital for Sick Children; 74, Harley street, Cavendish square.
- 1872 TOMES, CHARLES SISSMORE, M.A., F.R.S., 37, Cavendish square. C. 1887. *Lib. Com.* 1879.
- 1882 TOOTH, HOWARD HENRY, M.D., Demonstrator of Morbid Anatomy, St. Bartholomew's Hospital; Physician to the Metropolitan Hospital; Assistant Physician to the National Hospital for the Paralysed and Epileptic, Queen square; 34, Harley street, Cavendish square.
- 1879 TREVES, FREDERICK, Surgeon to, and Lecturer on Surgery at, the London Hospital; 6, Wimpole street, Cavendish square. C. 1895—. *Referee*, 1890—. *Sci. Com.* 1889—. *Trans.* 5.
- 1859 TRUMAN, EDWIN THOMAS, Surgeon-Dentist in Ordinary to Her Majesty's Household; 23, Old Burlington street.
- 1889 TURNBULL, GEORGE LINDSAY, M.B., Grove House, 76, Ladbroke grove.
- 1875 TURNER, FRANCIS CHARLEWOOD, M.D., Physician to the London Hospital; Consulting Physician to the North-Eastern Hospital for Children; 15, Finsbury square. C. 1895—.
- 1882 TURNER, GEORGE ROBERTSON, Visiting Surgeon to the Seamen's Hospital, Greenwich; Assistant Surgeon to, and Lecturer on Anatomy at, St. George's Hospital; 49, Green street, Park lane.

*Elected*

- 1894 **TURNER, PHILIP DYMOCK, M.D.**, 95, Cromwell Road.
- 1891 **TWEED, REGINALD, M.D.**, 55, Upper Brook street, Grosvenor square.
- 1892 **TWEEDY, JOHN**, Professor of Ophthalmic Medicine and Surgery in University College, Ophthalmic Surgeon to University College Hospital, and to the Royal London Ophthalmic Hospital; 100, Harley Street, Cavendish square.
- 1876 **VENN, ALBERT JOHN, M.D.**, Physician for the Diseases of Women, West London Hospital; 70A, Grosvenor street, and Hemnal Wood, Chislehurst.
- 1870 **VENNING, EDGCOMBE**, 30, Cadogan place.
- 1865 **VERNON, BOWATER JOHN**, Ophthalmic Surgeon to St. Bartholomew's Hospital and to the West London Hospital; 14, Clarges street, Piccadilly.
- 1867 **VINTRAS, ACHILLE, M.D.**, Physician to the French Embassy, and Senior Physician to the French Hospital and Dispensary, Shaftesbury Avenue; 19A, Hanover square.
- 1891 **VOELCKER, ARTHUR FRANCIS, M.D., B.S.**, Pathologist and Curator of the Museum, and Lecturer on Biology at the Middlesex Hospital; 31, Harley street, W.
- 1886 **WAINSWRIGHT, BENJAMIN, M.B., C.M.**, Assistant Surgeon to Charing Cross Hospital and to the Royal Westminster Ophthalmic Hospital; 67, Grosvenor street, Grosvenor square.
- 1884 **WAKLEY, THOMAS, jun.**, 5, Queen's Gate, South Kensington.
- 1883 **WALLER, AUGUSTUS, M.D., F.R.S.**, Lecturer on Physiology, St. Mary's Hospital; Weston Lodge, 16, Grove End road, St. John's Wood. *Referee*, 1895—.
- 1888 **WALLIS, FREDERICK CHARLES, M.B., B.C.**, Assistant Surgeon to the Charing Cross Hospital; 26, Welbeck street, Cavendish square.



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- 1873 WALSHAM, WILLIAM JOHNSON, C.M., Senior Assistant Surgeon to, and Lecturer on Anatomy at, St. Bartholomew's Hospital; Surgeon to the Metropolitan Free Hospital; 77, Harley street, Cavendish square. C. 1888-9. *Referee*, 1895—. *Lib. Com.* 1882-5. *Trans.* 7.
- 1886 WARD, ALLAN OGIER, M.D., Lansdowne House, High road, Tottenham.
- 1890 WARD, ARTHUR HENRY, Surgeon to Out-patients, Lock Hospital; 7, Hertford street, Mayfair.
- 1891 WARING, HOLBURN JACOB, M.B., B.S., B.Sc., 9, Upper Wimpole street.
- 1877 WARNER, FRANCIS, M.D., Physician to, and Lecturer on Materia Medica and Therapeutics at, the London Hospital; 5, Prince of Wales terrace, Kensington Palace, *Trans.* 1.
- 1889 WASHBOURN, JOHN WYCHENFORD, M.D., Assistant Physician to, Physician in Charge of Electrical Department, Joint Lecturer on Physiology, and Demonstrator of Bacteriology at, Guy's Hospital; Physician to the London Fever Hospital; 15, Trinity square, S.E. *Trans.* 1.
- 1894 WATERHOUSE, HERBERT FURNIVALL, C.M., Assistant Surgeon to the Charing Cross Hospital; 81, Wimpole street, W.
- †1861 WATSON, WILLIAM SPENCER, M.B., Surgeon to the Throat Department of the Great Northern Central Hospital; Surgeon to the Royal South London Ophthalmic Hospital; 7, Henrietta street, Cavendish square. C. 1883-4. *Trans.* 1.
- 1879 DE WATTEVILLE, ARMAND, M.D., 30, Welbeck street, Cavendish square.
- 1892 WEAVER, FREDERICK POYNTON, M.D., Cedar Lawn, Hampstead Heath.
- †1891 WEBER, FREDERIC PARKES, M.D., 19, Harley street, W.

*Elected*

- 1857 WEBER, HERMANN, M.D., Consulting Physician to the German Hospital; 10, Grosvenor street, Grosvenor square. C. 1874-5. V.P. 1885-6. *Sci. Com.* 1889—. *Referee*, 1869-73, 1878-84. *Lib. Com.* 1864-73 *Trans.* 6.
- 1895 WELLS, SYDNEY RUSSELL, M.B., 14, Girdler's road, West Kensington.
- †1854 WELLS, SIR THOMAS SPENCER, Bart., Hon. M.D. Dublin, Leyden, Bologna, Charkoff; Surgeon-in-Ordinary to H.M.'s Household; Consulting Surgeon to the Samaritan Free Hospital for Women and Children; Foreign Associate, Académie de Médecine, Paris; 3, Upper Grosvenor street. C. 1870. V.P. 1881. *Trans.* 14. *Pro.* 1.
- †1842 WEST, CHARLES, M.D., Foreign Associate, Académie de Médecine, Paris; 4, Evelyn Mansions, Carlyle place, Victoria street, S.W. C. 1855-6. V.P. 1863. P. 1877-8. *Referee*, 1848-54, 1857-62, 1864-76, 1880. *Sci. Com.* 1863. *Lib. Com.* 1844-7, 1851. *Trans.* 2.
- 1877 WEST, SAMUEL, M.D., Assistant Physician to St. Bartholomew's Hospital; Senior Physician to the Royal Free Hospital; 15, Wimpole street, Cavendish square. C. 1894. *Lib. Com.* 1892-4. *Trans.* 4.
- 1888 WETHERED, FRANK JOSEPH, M.D., Assistant Physician to the Hospital for Consumption, Brompton; 83, Harley street, Cavendish square. *Trans.* 1.
- 1881 WHARRY, ROBERT, M.D., 6, Gordon square.
- 1878 WHARTON, HENRY THORNTON, M.A., Senior Honorary Surgeon to the Kilburn Dispensary; "Madresfield," Acol road, Priory road, West Hampstead.
- 1875 WHIPHAM, THOMAS TILLYER, M.D., Physician to, and Lecturer on Medicine at, St. George's Hospital; 11, Grosvenor street, Grosvenor square. C. 1892-8.
- 1891 WHITE, CHARLES PERCIVAL, M.B., B.C., 144, Sloane street.

*Elected*

- 1881 WHITE, WILLIAM HALE, M.D., Physician to, and Lecturer on Materia Medica at, Guy's Hospital; 65, Harley street, Cavendish square. *Referee*, 1888—. *Trans.* 4.
- 1890 WHITE-COOPER, W. G. O., M.B., 5, Courtfield road, Gloucester road, S.W.
- 1877 WHITMORE, WILLIAM TICKLE, Senior Surgeon to the Westminster General Dispensary, to the St. George's and St. James's Dispensary, and to the Gordon Hospital for Diseases of the Rectum; 7, Arlington street, Piccadilly.
- 1863 WILKS, SAMUEL, M.D., LL.D., F.R.S., Physician in Ordinary to their Royal Highnesses the Duke and Duchess of Connaught; Consulting Physician to Guy's Hospital, and Member of the Senate of the University of London; 72, Grosvenor street, Grosvenor square. *Referee*, 1872-81. *Sci. Com.* 1.
- 1890 WILLCOCKS, FREDERICK, M.D., Physician to Out-Patients, and Lecturer on Materia Medica and Therapeutics, at the Charing Cross Hospital; Physician to the Evelina Hospital for Sick Children; 14, Mandeville place, Manchester square.
- †1865 WILLETT, ALFRED, *Trustee*; Surgeon to, and Lecturer on Surgery at, St. Bartholomew's Hospital; Surgeon to St. Luke's Hospital; 36, Wimpole street, Cavendish square. C. 1880-81. V.P. 1890-91. *Referee*, 1882-89, 1892—. *Bldg. Com.* 1889-92. *Ho. Com.* 1892—. *Trans.* 2.
- 1887 WILLETT, EDGAR, M.B., 25, Welbeck street, Cavendish square.
- 1888 WILLIAMS, CAMPBELL, 24, Welbeck street, Cavendish square.
- 1866 WILLIAMS, CHARLES THEODORE, M.A., M.D., *Trustee for Debenture-holders*; Consulting Physician to the Hospital for Consumption and Diseases of the Chest, Brompton; 2, Upper Brook street, Grosvenor square. C. 1884-5. *Referee*, 1888—. *Lib. Com.* 1880-3. *Sci. Com.* 1889—. *Trans.* 5.

*Elected*

- 1881 WILLIAMS, DAWSON, M.D., Physician to the East London Hospital for Children ; 101, Harley street. *Trans.* 1.
- 1872 WILLIAMS, SIR JOHN, Bart., M.D., Physician-Accoucheur to H.R.H. the Princess Beatrice ; Emeritus Professor of Obstetric Medicine, University College, London ; Consulting Obstetric Physician to University College Hospital ; 63, Brook street, Grosvenor square. C. 1891. *Referee*, 1878-90. *Lib. Com.* 1876-82.
- 1890 WILLS, WILLIAM ALFRED, M.D., Assistant Physician to the Westminster Hospital ; 23, Lower Seymour street, Portman square.
- 1879 WOAKES, EDWARD, M.D., Senior Aural Surgeon to the London Hospital ; 78, Harley street, Cavendish square.
- 1885 WOLFENDEN, RICHARD NORRIS, M.D., Physician to the Hospital for Diseases of the Throat, Golden square ; 19, Harley street, Cavendish square.
- 1887 WOOD, THOMAS OUTTERSON, M.D., 40, Margaret street, Cavendish square.
- 1891 WOODFORDE, ALFRED POWNALL, 160, Goldhawk road.
- 1892 WOODHEAD, GERMAN SIMS, M.D., Director of the Research Laboratory, Conjoint Board of R.C.P.Lond. and R.C.S.Eng. ; 1, Nightingale lane, Balham.
- 1890 WYNTER, WALTER ESSEX, M.D., Assistant Physician to the Middlesex Hospital ; 30, Upper Berkeley street, Portman square.

# LIST OF RESIDENT FELLOWS

ARRANGED ACCORDING TO  
DATE OF ELECTION

- |      |                                     |      |                                  |
|------|-------------------------------------|------|----------------------------------|
| 1838 | Henry Spencer Smith.                | 1857 | Henry Cooper Rose, M.D.          |
| 1840 | Sir James Paget, Bt., F.R.S.        |      | Henry Walter Kiallmark.          |
| 1841 | Paul Jackson.                       | 1858 | John William Ogle, M.D.          |
| 1842 | Sir John Simon, K.C.B., F.R.S.      | 1859 | Wm. Howship Dickinson, M.D.      |
|      | Charles West, M.D.                  |      | Edwin Thomas Truman.             |
|      | John Erichsen, F.R.S.               |      | Richard Barwell.                 |
| 1843 | Henry Lee.                          |      | Edward Tegar.                    |
| 1845 | George D. Pollock.                  |      | William E. Stewart.              |
|      | Sir Edwin Saunders.                 | 1860 | William Ogle, M.D.               |
|      | Edward U. Berry.                    |      | Thomas Bryant.                   |
| 1847 | Sir G. Johnson, M.D., F.R.S.        |      | John Couper.                     |
| 1848 | Sir Edward H. Sieveking, M.D.       |      | Henry Howard Hayward.            |
|      | John Clarke, M.D.                   | 1861 | William Spencer Watson.          |
| 1849 | C. H. F. Routh, M.D.                | 1862 | Lionel Smith Beale, M.B., F.R.S. |
| 1850 | Sir R. Quain, Bt., M.D., F.R.S.     |      | Edmund Symes Thompson, M.D.      |
| 1851 | John Birkett.                       |      | Reginald Edward Thompson, M.D.   |
|      | John A. Kingdon.                    |      | George Cowell.                   |
|      | Peter Y. Gowlland.                  | 1863 | J. L. H. Langdon-Down, M.D.      |
|      | Bernard E. Brodhurst.               |      | Samuel Wilks, M.D., F.R.S.       |
|      | Robert J. Spitta, M.D.              |      | Samuel Fenwick, M.D.             |
| 1852 | William Adams.                      |      | Julius Althaus, M.D.             |
|      | Sir Henry Thompson.                 |      | Sydney Ringer, M.D., F.R.S.      |
| 1853 | Robert Brudenell Carter.            |      | Thomas Smith.                    |
| 1854 | Sir Alfred B. Garrod, M.D., F.R.S.  |      | Arthur B. R. Myers.              |
|      | Sir Thomas Spencer Wells, Bt.       |      | William Sedgwick.                |
| 1855 | J. Russell Reynolds, M.D., F.R.S.   | 1864 | John Harley, M.D.                |
|      | William Marcet, M.D., F.R.S.        |      | Thomas William Nunn.             |
| 1856 | Charles J. Hare, M.D.               | 1865 | James Edward Pollock, M.D.       |
|      | William Bird.                       |      | Reginald Southey, M.D.           |
|      | Jonathan Hutchinson, F.R.S.         |      | George Fielding Blandford, M.D.  |
|      | Timothy Holmes.                     |      | Sir Dyce Duckworth, M.D.         |
|      | Alonzo H. Stocker, M.D.             |      | Frederick W. Pavy, M.D., F.R.S.  |
| 1857 | Sir William Overend Priestley, M.D. |      | John Langton.                    |
|      | George Harley, M.D., F.R.S.         |      | Frederick James Gant.            |
|      | Hermann Weber, M.D.                 |      | Alfred Willett.                  |

- 1865 Bowater John Vernon.  
Alfred Cooper.  
Christopher Heath.
- 1866 Thomas Fitz-Patrick, M.D.  
Samuel Jones Gee, M.D.  
Charles Theodore Williams, M.D.  
Heywood Smith, M.D.  
William Selby Church, M.D.
- 1867 William Henry Day, M.D.  
Achille Vintras, M.D.  
Richard Douglas Powell, M.D.  
F. Howard Marsh.  
Henry Power.  
Sir William MacCormac.  
Thomas Pickering Pick.  
Charles Arthur Aikin.
- 1868 H. Charlton Bastian, M.D., F.R.S.  
Sir W. H. Broadbent, Bart., M.D.  
Thomas Buzzard, M.D.  
John Cavafy, M.D.  
Walter Butler Cheadle, M.D.  
Sir Thos. Crawford, K.C.B., M.D.  
T. Henry Green, M.D.  
William Chapman Grigg, M.D.  
John Croft.  
George Eastes.
- 1869 Joseph Frank Payne, M.D.  
Arthur E. Sansom, M.D.  
Thomas Laurence Read.
- 1870 J. Warrington Haward.  
Edgecombe Venning.  
Clement Godson, M.D.  
Reginald Harrison.  
Robert Leamon Bowles, M.D.
- 1871 William Cayley, M.D.  
Charles Henry Ralfe, M.D.  
Thomas L. Brunton, M.D., F.R.S.  
J. Hughlings Jackson, M.D., F.R.S.  
Henry Sutherland, M.D.  
George Vivian Poore, M.D.  
Walter Rivington, M.S.  
Benjamin Duke.
- 1872 T. Gilbert Smith, M.D.  
George B. Brodie, M.D.  
Sir John Williams, Bart., M.D.  
Sir J. Fayrer, M.D., F.R.S.  
Charles S. Tones, M.A., F.R.S.  
Sir William Bartlett Dalby.
- 1873 William Miller Ord, M.D.  
Frederick Taylor, M.D.  
Norman Moore, M.D.  
John Curnow, M.D.  
William R. Gowers, M.D., F.R.S.  
Sir Wm. Guyer Hunter, M.D.
- 1873 Jeremiah McCarthy.  
Wm. Johnson Smith.  
Robert William Parker.  
Alex. O. MacKellar.  
Henry T. Butlin.  
Charles Higgins.  
William J. Walsham.
- 1874 Alfred Lewis Galabin, M.D.  
George Thin, M.D.  
Alfred B. Duffin, M.D.  
John Mitchell Bruce, M.D.  
Henry Morris.  
William Laidlaw Purves.  
William Harrison Cripps.  
Henry G. Howse, M.S.  
Herbert William Page.  
Frederic Durham.  
William Robert Smith, M.D.
- 1875 Thomas T. Whipham, M.B.  
Francis Charlewood Turner, M.D.  
Thomas Crawford Hayes, M.D.  
Charles Henry Carter, M.D.  
Waren Tay.  
Edmund J. Spitta.  
Samuel C. Osborn.  
Fletcher Beach, M.B.
- 1876 Thomas Barlow, M.D.  
Wm. Lewis Dudley, M.D.  
Albert J. Venn, M.D.  
John Knowsley Thornton, M.B.  
N. Charles Macnamara.  
John N. C. Davies-Colley, M.C.
- 1877 Felix Semon, M.D.  
Sidney Coupland, M.D.  
Francis Warner, M.D.  
William Ewart, M.D.  
Alfred Pearce Gould, M.S.  
Rickman J. Godlee, M.S.  
Alban H. G. Doran.  
George Ernest Herman, M.B.  
Samuel West, M.D.  
John Abercrombie, M.D.  
George Allan Heron, M.D.  
Joseph A. Ormerod, M.D.  
P. Henry Pye-Smith, M.D., F.R.S.  
Edward Nettleship.  
William Henry Bennett.  
William T. Whitmore.
- 1878 Sir Jas. Crichton-Browne, M.D.  
Fred. T. Roberts, M.D.  
Sir Joseph Lister, Bart., F.R.S.  
Clinton T. Dent.  
John H. Morgan.  
Donald W. Charles Hood, M.D.

- 1878 Henry Gervis, M.D.  
Henry Thornton Wharton.
- 1879 Edward Woakes, M.D.  
Armand de Watteville, M.D.  
Malcolm A. Morris.  
A. E. Cumberbatch.  
Edmund Owen.  
Arthur E. J. Barker.  
Frederick Treves.  
Horatio Donkin, M.D.  
Thomas John MacLagan, M.D.  
Andrew Clark.  
Francis Henry Champneys, M.D.  
William Watson Cheyne, F.R.S.  
George Henry Savage, M.D.  
H. H. Clutton, M.A.  
Frederic S. Eve.  
E. Noble Smith.  
William Henry Allchin, M.D.  
F. G. Dawtrey Drewitt, M.D.
- 1880 Robert Alex. Gibbons, M.D.  
David Ferrier, M.D., F.R.S.  
Vincent Dormer Harris, M.D.  
Edmund Distin Maddick.  
Jas. John MacWhirter Dunbar, M.D.  
James William Browne, M.B.  
William Appleton Meredith, M.B.  
Malcolm Macdonald McHardy.  
A. Boyce Barrow.  
William Murrell, M.D.  
Leslie Ogilvie, M.B.  
George Ogilvie, M.B.  
Charles Edward Beevor, M.D.  
Thomas Colcott Fox, M.B.  
George Henry Makins.
- 1881 Francis de Havilland Hall, M.D.  
Robert Wharry, M.D.  
Cecil Yates Biss, M.D.  
Richard Clement Lucas, B.S.  
Stephen Mackenzie, M.D.  
William Hale White, M.D.  
Eustace Smith, M.D.  
Percy Kidd, M.D.  
Oswald A. Browne.  
W. Bruce Clarke, M.B.  
Dawson Williams, M.D.  
George Lindsay Johnson, M.D.  
Henry Edward Juler.  
Jonathan F. C. H. Macready.  
C. B. Lockwood.
- 1882 Philip J. Hensley, M.D.  
Ernest Clarke, M.D.  
John Barclay Scriven.  
George Robertson Turner.
- 1882 Howard Henry Tooth, M.D.  
Herbert Isambard Owen, M.D.  
Charles R. B. Kestley.  
Anthony A. Bowlby.  
Amand J. McC. Routh, M.D.  
Seymour J. Sharkey, M.D.  
William Lang.  
Henry Radcliffe Crocker, M.D.
- 1883 Edwin Clifford Beale, M.A., M.B.  
James Kingston Fowler, M.D.  
James Frederic Goodhart, M.D.  
John Charles Galton, M.A.  
W. Hamilton A. Jacobson, M.Ch.  
Walter H. Jessop, M.B.  
Walter Edmunds, M.C.  
Victor A. Horsley, F.R.S.  
Dudley Wilmot Buxton, M.D.  
Charles Douglas F. Phillips, M.D.  
John James Pringle, M.B.  
Henry Roxburgh Fuller, M.D.  
Wilmot Parker Herringham, M.D.  
Augustus Waller, M.D.  
William Pasteur, M.D.  
Edward Albert Schäfer, F.R.S.  
John Bland Sutton.  
William Rose, M.B.  
Storer Bennett.  
Robert Marcus Gunn, M.B.  
James Dixon Bradshaw, M.B.
- 1884 George Newton Pitt, M.D.  
Charles Stonham.  
Stanley Boyd, M.B.  
William Arbuthnot Lane, M.S.  
Arthur Marmaduke Sheild, M.B.  
Frederic Bowreman Jessett.  
Sidney Harris Cox Martin, M.D.  
George Lawson.  
Thomas Wakley, jun.  
F. Swinford Edwards.  
James Johnston, M.D.  
William Duncan, M.D.  
Charles Chinner Fuller.  
Jean Samuel Keser, M.D.  
George Richard Turner Phillips.  
Bilton Pollard.
- 1885 Alexander Haig, M.D.  
Theodore Dyke Acland, M.D.  
Frederick Walker Mott, M.D.  
James Berry.  
John Cahill.  
John Poland.  
Heinrich Port, M.D.  
R. Norris Wolfenden, M.D.  
A. C. Butler-Smythe.

- 1885 Charles Alfred Ballance, M.S.  
Walter S. A. Griffith, M.D.  
John Edward Squire, M.D.  
John D. Malcolm, M.B., C.M.  
Phineas S. Abraham, M.D.  
Henry Willingham Gell, M.B.
- 1886 Robert Maguire, M.D.  
Harrington Sainsbury, M.D.  
Cuthbert Hilton Golding-Bird, M.B.  
Benjamin Wainewright, M.B., C.M.  
Charles E. Leopold B. Hudson.  
Lauriston Elgie Shaw, M.D.  
Charters James Symonds, M.S.  
Robert Boxall, M.D.  
Allan Ogier Ward, M.D.  
Archibald Edward Garrod, M.D.  
Stephen Paget.  
William Radford Dakin, M.D.  
Samuel Herbert Habershon, M.D.  
Arthur Quarry Silcock.  
Arthur H. N. Lewers, M.D.
- 1887 Walter George Spencer.  
Thomas Outterson Wood, M.D.  
Edgar William Willett, M.B.  
Henry Lewis Jones, M.D.  
Francis George Penrose, M.D.  
Hugh Percy Dunn.  
Frederic William Hewitt, M.D.  
Harry Scott, M.D.  
James Barry Ball, M.D.  
Gilbert Richardson, M.D.  
D'Arcy Power, M.B.  
John Gay.  
James Calvert, M.D.  
Percy J. F. Lush, M.B.
- 1888 Robert Henry Scanes Spicer, M.D.  
Jonathan Hutchinson, jun.  
Campbell Williams.  
James Donelan, M.B., C.M.  
John Anderson, M.D., C.I.E.  
Laurie Asher Lawrence.  
Charles Arkle, M.D.  
Arthur Pearson Luff, M.D., B.Sc.  
Albert Carless, M.B., B.S.  
Frederick C. Wallis, M.B., B.C.  
Charles James Cullingworth, M.D.  
Edmund Cautley, M.D., B.C.  
H. Montague Murray, M.D.  
Arthur Symons Eccles, M.B.  
Frank Joseph Wethered, M.D.  
Edmund Wilkinson Roughton, M.D.  
Frederick William Cock, M.D.  
John Phillips, M.B.
- 1889 Montagu Handfield-Jones, M.D.
- 1889 Norman M. MacLehose, M.B.  
David Henry Goodsell.  
Raymond Johnson, M.B.  
John Fletcher Little, M.B.  
Henry Work Dodd.  
George Lindsay Turnbull, M.B.  
Sir William Roberts, M.D., F.R.S.  
Sidney Phillips, M.D.  
William Charles Bull, M.B.  
George P. Field.  
John Wychenford Washbourn, M.D.  
Charles Henry Cosens.  
Henry Percy Dean, M.B., M.S.  
Alfred Samuel Gubb.  
William Hunter, M.D.  
J. Inglis Parsons, M.D.  
Bernard Pitts, M.B., M.C.  
Robert Percy Smith, M.D., B.S.  
Herbert R. Spencer, M.D., B.S.  
Nestor Isidore Chas. Tirard, M.D.  
F. W. Humphery, M.D.
- 1890 John Rose Bradford, M.D., F.R.S.  
Roland Danvers Brinton, M.D.  
James Cagney, M.D.  
Charles D. B. Hale, M.D.  
Edwin Cooper Perry, M.D.  
Morton Smale.  
Frederick Willcocks, M.D.  
R. Ashton Rostock.  
William T. Holmes Spicer, M.B.  
Thomas Henry Crowle.  
Henry Walter Syers, M.D.  
Seymour Taylor, M.D.  
William Alfred Wills, M.D.  
G. O. White-Cooper, M.B.  
Herbert William Allingham.  
William Anderson.  
William A. F. Bateman.  
James Jackson Clarke, M.B.  
Leonard G. Guthrie, M.B., B.S.  
G. William Hill, M.D., B.Sc.  
Edward Law, M.D., C.M.  
Patrick Manson, M.D., C.M.  
William Wallis Ord, M.D.  
Humphry D. Rolleston, M.D., B.C.  
Arthur Henry Ward.  
Walter Essex Wynter, M.D., B.S.
- 1891 William Lee Dickinson, M.D.  
Herbert P. Hawkins, M.D., B.C.  
Cyril Ogle, M.A., M.B.  
Leonard Remfry, M.D.  
Arthur F. Voelcker, M.D., B.S.  
Alfred Pownall Woodforde.  
Charles Gordon Brodie.



- 1891 Herbert T. Herring, M.B., B.S.  
 Ernest Muirhead Little.  
 Henry Charrington Martin, M.D.  
 Launcelot Andrews, M.B.  
 Frederick William Andrewes, M.B.  
 Alfred Eddowes, M.D.  
 Herbert Morley Fletcher, M.D.  
 William Heaton Hamer, M.D.  
 William Bromfield Paterson.  
 Reginald Tweed, M.D.  
 Holburt Jacob Waring.  
 Frederic Parkes Weber, M.D.  
 F. E. Batten, M.B.  
 Thomas Jessopp Bokenham.  
 Henry Johnstone Campbell, M.D.  
 Norman Dalton, M.D.  
 P. R. W. De Santi.  
 P. W. Dove.  
 William J. Gow, M.D.  
 Charles Arthur Mercier, M.B.  
 Paul Frank Moline, M.B.  
 Edward Percy Paton, M.D.  
 Arthur Bowen Rendel, M.B., B.C.  
 M. Armand Ruffer, M.D.  
 James Samuel Risien Russell, M.B.  
 George Cockburn Smith.  
 Charles Percival White, M.B., B.C.  
 W. Page May, M.D.  
 Richard J. Reece.
- 1892 Edward Cotterell.  
 J. Dundas Grant, M.D.  
 R. J. Bliss Howard, M.D.  
 Thomas Horrocks Openshaw, M.B.  
 Henry Marmaduke Page.  
 William Bezly Thorne, M.D.  
 German Sims Woodhead, M.D.  
 W. H. Russell Forsbrook, M.D.  
 John Harold.  
 William Ward Leadam, M.D.  
 John Alfred Masters, M.D.  
 Gustave Schorstein, M.B.  
 Charles Sempill de Segundo.  
 John Tweedy.  
 E. H. Myddelton-Gavey.  
 E. Matthews James.
- 1892 J. S. Selwyn-Harvey, M.D.  
 St. Clair Thomson, M.D.  
 F. Manley B. Sims.  
 Solomon Charles Smith, M.D.  
 F. Poynton Weaver, M.D.  
 Henry Rayner, M.D.
- 1893 William Gordon, M.B.  
 James Taylor, M.D.  
 Howard Barrett.  
 Robert Cozens Bailey, M.B.  
 Henry Albert Caley, M.D.  
 Arthur Edward Giles, M.D.  
 Miles Miley, M.B.  
 Alfred A. Kanthack, M.D.  
 Kenneth McLeod, M.D.  
 D. Watkin Roberts, M.D.  
 Leonard A. Bidwell.  
 Frédéric F. Burghard, M.D, M.S.  
 William McAdam Eccles, M.B.  
 Vaughan Harley, M.D.  
 George Herschell, M.D.  
 Arnold Lawson.  
 Walter Knowsley Sibley, M.D.  
 Richard Sisley, M.D.  
 Henry Moore Bowman, M.D.  
 Richard Gill.  
 Joseph Sefton Sewill.  
 Thomas Vincent Dickinson, M.D.  
 Herbert Edward Durham, M.B.  
 Alexander Morison, M.D.  
 L. Hemington Pegler, M.D.  
 Herbt. Furnivall Waterhouse, M.D.  
 Philip D. Turner, M.D.
- 1894 Percy Furnivall.  
 R. L. Langdon-Down, M.B., B.C.  
 Allan Macfadyen, M.D., B.S.  
 Ernst Michels, M.D.  
 Wm. Rivers Pollock, M.B., B.C.  
 Thomas R. Smith, M.B., B.C.  
 Charles Slater, M.B.
- 1895 Charles Arthur Parker.  
 Sydney Russell Wells, M.B.  
 Alfred Milne Gossage, M.B.  
 Robert Murray Leslie, M.B.

## NON-RESIDENT FELLOWS

### *Elected*

- \*1851 *ACLAND, SIR HENRY W.*, Bart., K.C.B., M.D., LL.D., F.R.S., Honorary Physician to H.R.H. the Prince of Wales; late Regius Professor of Medicine in the University of Oxford; Oxford.
- 1839 *ALCOCK, SIR RUTHERFORD*, K.C.B., K.C.T., K.T.S., D.C.L., late H.M.'s Envoy Extraordinary at the Court of Pekin. *Trans.* 1.
- 1866 *ALLBUTT, THOMAS OLIFFORD*, A.M., M.D., LL.D. Glasgow, F.R.S., Regius Professor of Physic, Univ. Camb.; Consulting Physician to the Leeds General Infirmary; St. Rhadegund's, Cambridge. *Trans.* 3.
- 1884 *ANDERSON, ALEXANDER RICHARD*, Surgeon to the General Hospital, 5, East Circus Street, Nottingham. *Trans.* 1.
- 1862 *ANDREW, JAMES*, M.D., Branksome avenue, West Bournemouth. S. 1878-9. C. 1881-2. V.P. 1888. *Trans.* 1.
- \*1880 *APPLETON, HENRY*, M.D., 2, Swinburn villas, Romford, Essex.
- 1891 *BAKER, CHARLES ERNEST*, M.B., Marlborough road, St. Albans.
- \*1873 *BAKER, J. WRIGHT*, Consulting Surgeon to the Derbyshire General Infirmary [care of Dr. Benthall, 101, Friar gate, Derby].
- 1891 *BALGARNIE, WILFRED*, M.B., The Dutch House, Hartley Wintney, Winchfield.
- †1848 *BALLARD, EDWARD*, M.D., F.R.S., late Inspector, Medical Department, Local Government Board; 6, Ravenscroft Park, High Barnet, Herts. C. 1872. V.P. 1875-6. *Sci. Com.* 1889—. *Referee*, 1853-71. *Lib. Com.* 1855. *Trans.* 5.

*Elected*

- \*1866 *BANKS, SIR JOHN*, M.D., LL.D., D.Sc., K.C.B., Physician in Ordinary to the Queen in Ireland; Physician to Richmond, Whitworth, and Hardwicke Hospitals; Consulting Physician to Sir Patrick Dunn's and City of Dublin Hospitals; Regius Professor of Physic in the University of Dublin; Member of the Senate of the Royal University in Ireland; 45, Merrion square, Dublin.
- 1886 *BANKS, WILLIAM MITCHELL*, M.D., Surgeon to the Liverpool Royal Infirmary 28, Rodney street, Liverpool.
- 1882 *BARKER, FREDERICK CHARLES*, M.D., Surgeon-Major, Bombay Medical Service.
- \*1881 *BARNES, HENRY*, M.D., F.R.S. Ed., Physician to the Cumberland Infirmary; 6, Portland square, Carlisle.
- †1861 *BAERNES, ROBERT*, M.D., Liss, Hants. C. 1877-8. V.P. 1889-90. *Referee*, 1867-76, 1891—. *Lib. Com.* 1869-73. *Sci. Com.* 1889—. *Trans.* 4.
- 1840 *BARROW, BENJAMIN*, Consulting Surgeon to the Royal Isle of Wight Infirmary; Southlands Park road, Ryde.
- \*1860 *BEALEY, ADAM*, M.D., M.A., Filsham Lodge, Filsham road, St. Leonard's-on-Sea, Sussex.
- 1856 *BEARDSLEY, AMOS*, F.L.S., Bay villa, Grange-over-Sands, Lancashire.
- 1880 *BENNETT, ALEXANDER HUGHES*, M.D. (Travelling).
- 1889 *BENTLEY, ARTHUR J. M.*, M.D., Mena House, Pyramids, Cairo, Egypt.
- 1872 *BEVERLEY, MICHAEL*, M.D., Surgeon to the Norfolk and Norwich Hospital; 54, Prince of Wales road, Norwich.
- \*1865 *BICKERSTETH, EDWARD ROBERT*, Consulting Surgeon to the Liverpool Royal Infirmary; 2, Rodney street, Liverpool. *Trans.* 1.
- 1892 *BICKERSTETH, ROBERT ALEXANDER*, M.A., M.B., Assistant Surgeon to the Liverpool Royal Infirmary; 2, Rodney street, Liverpool.
- 1878 *BINDON, WILLIAM JOHN VEREKEE*, M.D., 48, St. Ann's street, Manchester.

*Elected*

- †1849 *BIRKETT, EDMUND LLOYD*, M.D., Consulting Physician to the City of London Hospital for Diseases of the Chest; Westbourne Rectory, Emsworth, Hampshire. C. 1865-6. *Referee*, 1851-9.
- 1865 *BLANCHET, HILARION*, Examiner to the College of Physicians and Surgeons, Lower Canada; 35, Conillard street, Quebec, Canada.
- 1869 *BOURNE, WALTER*, M.D. (Travelling).
- 1874 *BRADSHAW, A. F.*, C.B., Surgeon Major-General, Principal Medical Officer, H.M.'s Forces in India; Simla, India. [Agents: Holt & Co., 17, Whitehall place.]
- \*1867 *BRETT, ALFRED T.*, M.D., Watford House, Watford, Herts.
- 1876 *BRIDGES, ROBERT*, M.B., Manor House, Yattendon, Newbury, Berks.
- 1867 *BRIDGEWATER, THOMAS*, M.B., LL.D., Harrow-on-the-Hill, Middlesex.
- 1892 *BRONNER, ADOLPH*, M.D., 33, Manor row, Bradford.
- 1894 *BROOK, WILLIAM HENRY BREEFFIT*, 1, James street, Lincoln.
- 1888 *BROWNE, HENRY LANGLEY*, Moor House, West Bromwich.
- 1881 *BROWNE, JOHN WALTON*, M.D., Surgeon to the Belfast Royal Hospital; Surgeon to the Belfast Ophthalmic Hospital; 10, College square N., Belfast.
- 1864 *BUCKLE, FLEETWOOD*, M.D., Merton Lodge, Merton road, Southsea.
- 1871 *BUTT, WILLIAM F.*
- \*1851 *CADGE, WILLIAM*, Consulting Surgeon to the Norfolk and Norwich Hospital; 49, St. Giles's street, Norwich. *Trans.* 1.
- 1888 *CARTER, WILLIAM JEFFREYS BECHER*, Aliwal North, Cape Colony.
- 1884 *CHAPPEY, WAYLAND CHARLES*, M.D., Physician to the Royal Alexandra Hospital for Children; 13, Montpellier road, Brighton.

*Elected*

- 1859 CHANCE, FRANK, M.B., Burleigh House, Sydenham hill.
- 1885 CHAPMAN, PAUL MORGAN, M.D., Physician to the Hereford General Infirmary, 1, St. John street, Hereford. *Trans.* 1.
- \*1881 CHAVASSE, THOMAS FREDERICK, M.D., C.M., Surgeon to the Birmingham General Hospital; Consulting Surgeon to the Bromsgrove Hospital; 22, Temple row, Birmingham. *Trans.* 3.
- 1890 CHILDS, CHRISTOPHER, M.D., 2, Royal terrace, Weymouth.
- \*1873 CHISHOLM, EDWIN, M.D., Abergeldie, Ashfield, near Sydney, New South Wales.
- 1892 CLARK, JAMES CHARLES, Croft House, Margate road, Southsea.
- 1888 CLARKE, ROBERT HENRY, M.B., Westwood, Isle of Thanet, Kent.
- 1857 COATES, CHARLES, M.D., Consulting Physician to the Bath Royal United Hospital; 10, Circus, Bath.
- 1868 COCKLE, JOHN, A.M., M.D., F.L.S., Consulting Physician to the Royal Free Hospital; The Lodge, West Molesey. *Trans.* 2.
- 1893 COLE, ROBERT HENRY, M.B., Moorcroft, Hillingdon, Uxbridge.
- 1891 COOK, HERBERT GEORGE, M.D., B.S., 22, Newport road, Cardiff.
- \*1860 CORRY, THOMAS CHARLES STEUART, M.D., Ormeau terrace, and 1, Glenfield place, Belfast.
- 1891 COUMBE, JOHN BATTEN, M.D., Rosslyn, Clevedon, Somerset.
- 1894 CRAWFORD, WILLIAM SMYTH, 77, Mount Pleasant, Liverpool.
- \*1869 CRESSWELL, PEARSON R., Surgeon to the Merthyr General Hospital; Dowlais, Merthyr Tydvil.
- 1892 CROSS, FRANCIS RICHARDSON, M.B., Ophthalmic Surgeon to the Bristol Royal Infirmary, and Surgeon to the Bristol Eye Hospital; Worcester House, Clifton, Bristol.

*Elected*

- 1895 *DARDEL, JEAN*, M.D., Aix-les-Bains, Savoy.
- 1879 *DARWIN, FRANCIS*, M.B., F.R.S., Wychfield, Huntingdon road, Cambridge.
- 1874 *DAVIDSON, ALEXANDER*, M.D., Physician to the Liverpool Royal Infirmary; 2, Gambier terrace, Liverpool.
- †1878 *DAVY, RICHARD*, Consulting Surgeon to the Westminster Hospital; Burstone House, Bow, North Devon. *Trans.* 1.
- \*1882 *DAWSON, YELVERTON*, M.D., Heathlands, Southbourne-on-Sea, Hants.
- 1889 *DELÉPINE, SHERIDAN*, B.S., M.B., Professor of Pathology, Owens College, Manchester. *Trans.* 1.
- †1839 *DIXON, JAMES*, Consulting Surgeon to the Royal London Ophthalmic Hospital, Moorfields; Harrow Lands, Dorking. L. 1849-55. V.P. 1857-8. T. 1863-4. C. 1866-7. *Referee*, 1865. *Lib. Com.* 1845-8. *Trans.* 4.
- 1867 *DRAGE, CHARLES*, M.D., Hatfield, Herts.
- 1884 *DRAGE, LOVELL*, M.A., M.B., B.Ch.Oxon., Burleigh Mead, Hatfield, Herts.
- 1885 *DRUMMOND, DAVID*, M.D., 7, Saville place, Newcastle-on-Tyne.
- 1880 *DRURY, CHARLES DENNIS HILL*, M.D., Bondgate, Darlington.
- 1893 *DRYSDALE, J. H.*, 17, Rodney street, Liverpool.
- \*1871 *DUKES, CLEMENT*, M.D., B.S., Physician to Rugby School, and Senior Physician to the Hospital of St. Cross, Rugby; Sunnyside, Rugby, Warwickshire.
- 1867 *DUKES, MAJOR CHARLES*, M.D., Clarence Villa, Torrs park, Ilfracombe.
- \*1889 *DUNCAN, JOHN*, M.D., St. Petersburg, Russia.
- 1843 *DURRANT, CHRISTOPHER MERCER*, M.D., Consulting Physician to the East Suffolk and Ipswich Hospital; Northgate street, Ipswich, Suffolk.
- 1872 *EAGER, REGINALD*, M.D., Northwoods, near Bristol.

*Elected*

- 1887 *EASMON, JOHN FARRELL*, M.D., Assistant Colonial Surgeon, Gold Coast Colony, and Acting Chief Medical Officer of the Colony ; Accra, Gold Coast, West Africa.
- 1887 *ELLIOTT, JOHN*, Whitefriars Lodge, Chester.
- 1848 *ELLIS, GEORGE VINEY*, Minsterworth, Gloucester. C. 1863-4. *Trans.* 2.
- 1868 *ELLIS, JAMES*, M.D., The Sanatorium, Anaheim, Los Angeles County, California.
- \*1854 *ELLISON, JAMES*, M.D., Surgeon-in-Ordinary to the Royal Household, Windsor ; 14, High street, Windsor.
- 1889 *ELLISTON, WILLIAM ALFRED*, M.D., Stoke Hall, Ipswich.
- \*1875 *FAGAN, JOHN*, Surgeon to, and Lecturer on Clinical Surgery at, the Belfast Royal Hospital ; 19, Great Victoria street, Belfast.
- 1869 *FAIRBANK, FREDERICK ROYSTON*, M.D., 16, Eversfield place, St. Leonard's-on-Sea.
- 1887 *FEBNY, MICHAEL HENRY*, Les Avants, Montreux, Switzerland.
- \*1872 *FENWICK, JOHN C. J.*, M.D., Physician to the Durham County Hospital ; Long Framlington, Morpeth.
- 1879 *FINLAY, DAVID WHITE*, M.D., Professor of the Practice of Medicine in the University of Aberdeen ; Physician to the Aberdeen Royal Infirmary ; Consulting Physician to the Royal Hospital for Diseases of the Chest, London ; 2, Queen's terrace, Aberdeen. *Referee*, 1891-3. *Trans.* 2.
- 1842 *FLETCHER, THOMAS BELL ELCOCK*, M.D., Consulting Physician to the Birmingham General Hospital ; 8, Clarendon crescent, Leamington. *Trans.* 1.
- \*1864 *FOLKER, WILLIAM HENRY*, Consulting and late Hon. Surgeon to the North Staffordshire Infirmary ; Bedford House, Hanley, Staffordshire.
- 1892 *FOSTER, MICHAEL GEORGE*, M.A., M.B., Great Shelford, Cambridge.
- 1859 *FOX, EDWARD LONG*, M.D., Consulting Physician to the Bristol Royal Infirmary ; Church House, Clifton, Gloucestershire.

*Elected*

- 1871 *FRANK, PHILIP*, M.D., Cannes, France.
- \*1884 *FRANKS, KENDAL*, M.D., Surgeon to the Adelaide Hospital and to the Throat and Ear Hospital, Dublin ; Surgeon in Ordinary to the Lord Lieutenant ; 6, Fitzwilliam square, Dublin. *Trans.* 2.
- \*1889 *FREEMAN, HENRY WILLIAM*, 24, The Circus, Bath.
- 1876 *FURNER, WILLOUGHBY*, M.D., Surgeon to the Sussex County Hospital ; Brunswick square, Brighton.
- \*1864 *GAIRDNER, WILLIAM TENNANT*, M.D., LL.D., F.R.S., Physician in Ordinary to H.M. the Queen in Scotland ; Professor of the Practice of Medicine in the University of Glasgow ; Physician to the Western Infirmary, Glasgow ; 225, St. Vincent street, Glasgow. *Trans.* 1.
- 1885 *GAMGEE, ARTHUR*, M.D., F.R.S., Montreux, Switzerland.
- 1867 *GARLAND, EDWARD CHARLES*, Yeovil, Somerset.
- 1867 *GARLIKE, THOMAS W.*, Malvern Cottage, Churchfield road, Ealing.
- 1879 *GARSTANG, THOMAS WALTER HARROP*, Headingley House, Knutsford, Cheshire.
- \*1889 *GASKELL, WALTER HOLBROOK*, M.D., F.R.S., Lecturer on Physiology, University of Cambridge ; The Uplands, Great Shelford, Cambs.
- 1884 *GIBBES, HENEAGE*, M.D., Professor of Pathology in the University of Michigan ; Ann Arbor, Michigan, U.S.A.
- \*1890 *GORDON, WILLIAM*, M.D., Barnfield Lodge, Exeter.
- 1889 *GREENE, GEORGE EDWARD JOSEPH*, Monte Vista, Ferns, County Wexford.
- \*1875 *GREENFIELD, WILLIAM SMITH*, M.D., Professor of General Pathology in the University of Edinburgh ; 7, Heriot row, Edinburgh. *Sci. Com.* 1879. *Referee*, 1881.
- 1882 *GRESSWELL, DAN ASTLEY*, M.A., M.D., D.P.H., Melbourne, Victoria.



*Elected*

- 1889 *GRIFFITHS, JOSEPH*, M.A., M.D., C.M., Assistant to the Professor of Surgery in the University of Cambridge; 4, King's parade, Cambridge.
- 1852 *GROVE, JOHN*, Pitt House, 15, Johnstown street, Bath.
- 1870 *HAMILTON, ROBERT*, Consulting Surgeon to the Royal Southern Hospital, Liverpool; Magheraybuoy, Portrush, Co. Antrim, Ireland.
- 1892 *HARSANT, WILLIAM HENRY*, 16, Pembroke road, Clifton, Bristol.
- 1890 *HAVILAND, FRANK PAPILLON*, M.B., B.C., 57, Warrior square, St. Leonard's-on-Sea.
- 1885 *HAWKINS, FRANCIS HENRY*, M.B., Physician to the Royal Berkshire Hospital; 26, Portland place, Reading.
- 1861 *HAYWARD, WILLIAM HENRY*, Oxford road, Burnley, Lancashire.
- †1843 *HOLDEN, LUTHER*, Consulting Surgeon to St. Bartholomew's Hospital, to the Metropolitan Dispensary, and to the Foundling Hospital; Pinetoft, Ipswich. C. 1859. L. 1865. V.P. 1874. *Referee*, 1866-7. *Lib. Com.* 1858.
- 1894 *HOLLAND, JAMES FRANK*, M.D., St. Moritz, Engadine, Switzerland.
- 1868 *HOLLIS, WILLIAM AINSLIE*, M.A., M.D., Physician to the Sussex County Hospital; 8, Cambridge road, Brighton. *Trans.* 1.
- †1846 *HOLTHOUSE, CARSTEN*, 1, Bath terrace, Richmond. C. 1863. *Referee*, 1870-6. *Lib. Com.* 1859-60.
- 1865 *HOWARD, BENJAMIN*, M.D. [New York, U.S.A.] *Trans.* 1.
- 1881 *HOWARD, HENRY*, M.B., Medical Officer of Health, Williamstown, Melbourne, Victoria.
- 1855 *HUMPHRY, SIR GEORGE MURRAY*, M.D., D.Sc., LL.D., F.R.S., Surgeon to Addenbrooke's Hospital; Professor of Surgery in the University of Cambridge. *Trans.* 9.
- 1882 *HUMPHRY, LAURENCE*, M.D., 3, Trinity street, Cambridge.

*Elected*

- 1849 *HUSSEY, EDWARD LAW*, 24, Winchester road, Oxford.  
*Trans.* 1.
- 1847 *IMAGE, WILLIAM EDMUND*, Herringswell House, Mildenhall, Suffolk. *Trans.* 1.
- 1863 *JACKSON, THOMAS VINCENT*, Senior Surgeon to the Wolverhampton and Staffordshire General Hospital; Whetstone House, Waterloo road south, Wolverhampton.
- \*1883 *JENKINS, EDWARD JOHNSTONE*, M.D., The Australian Club, Sydney, New South Wales.
- †1851 *JENNER, SIR WILLIAM*, Bart., M.D., G.C.B., D.C.L., LL.D.Cantab., LL.D.Edin., F.R.S., Physician in Ordinary to H.M. the Queen, and to H.R.H. the Prince of Wales; Emeritus Professor of Clinical Medicine in University College, London; and Consulting Physician to University College Hospital; Greenwood, Bishop's Waltham, Hants. C. 1864. V.P. 1875. *Referee*, 1855, 1859-63. *Trans.* 3.
- 1881 *JENNINGS, WILLIAM OSCAR*, M.D., 35, Rue Marbœuf, Avenue des Champs-Élysées, Paris.
- 1851 *JOHNSON, EDMUND CHARLES*, Corresponding Member of the Medical and Philosophical Society of Florence, and of "l'Institut Gènevois."
- 1889 *JOHNSON, HAROLD J.*, Senior Assistant, Gloucester County Asylum, Gloucester.
- 1848 *JOHNSTONE, ATHOL ARCHIBALD WOOD*, Consulting Surgeon to the Royal Alexandra Hospital for Sick Children, St. Moritz House, 61, Dyke road, Brighton. *Lib. Com.* 1860. *Trans.* 1.
- 1876 *JONES, LESLIE HUDSON*, M.D., Limefield House, Cheetham hill, Manchester.
- \*1875 *JONES, PHILIP SYDNEY*, M.D., Consulting Surgeon to the Sydney Infirmary; Examiner in Medicine, and Fellow of the Senate, Sydney University; 10, College street, Sydney, New South Wales. [Agents: Messrs. D. Jones & Co., Wool Exchange, Coleman Street, E.C.]

*Elected*

- 1865 *JORDAN, FURNEAUX*, Consulting Surgeon to the Queen's Hospital, Birmingham; Selly Hill, Birmingham.
- 1872 *KELLY, CHARLES*, M.D., Professor of Hygiene in King's College, London; Medical Officer of Health for the West Sussex Combined Sanitary District; Ellesmere, Gratwicke road, Worthing, Sussex.
- \*1848 *KENDELL, DANIEL BURTON*, M.B., Thornhill House, Walton, near Wakefield, Yorkshire.
- \*1890 *KERR, J. G. DOUGLAS*, M.B., C.M., 6, The Circus, Bath.
- \*1877 *KHORY, RUSTOMJEE NASERWANJEE*, M.D., Honorary Obstetric Physician to the Bai Motlibari and Sir Dinsha Petit Hospitals, &c.; Hormazd Villa, Khumballa hill, Bombay.
- 1883 *KNAPTON, GEORGE*, Queen's square, Blackpool.
- 1888 *KYNSEY, WILLIAM RAYMOND*, C.M.G., Inspector-General of Hospitals, Colombo, Ceylon.
- 1889 *LANCASTER, ERNEST LE CRONIER*, M.B., B.Ch., Assistant Physician to the Swansea Hospital; Winchester House, Swansea, S. Wales.
- 1891 *LANE, HUGH*, 11, The Circus, Bath.
- \*1873 *LARCHER, O.*, M.D., Laureate of the Institute of France, of the Medical Faculty, and Academy of Paris, &c.; 97, Rue de Passy, Passy, Paris.
- 1862 *LATHAM, PETER WALLWORK*, M.A., M.D., late Downing Professor of Medicine, Cambridge University; Senior Physician to Addenbrooke's Hospital, Cambridge; 17, Trumpington street, Cambridge.
- \*1890 *LAWRIE, EDWARD*, M.B., Surgeon Lieutenant-Colonel, Indian Medical Department; Residency Surgeon; Hyderabad, Deccan.
- 1880 *LAYCOCK, GEORGE LOCKWOOD*, M.B., C.M., Melbourne, Victoria, Australia.
- 1892 *LAZARUS-BARLOW, WALTER SYDNEY*, M.B., The Acacias, Chesterton, Cambridge. *Sci. Com.* 1892—.
- \*1886 *LEDIARD, HENRY AMBROSE*, M.D., Surgeon to the Cumberland Infirmary; 35, Lowther street, Carlisle.

*Elected*

- 1882 *LEDWICH, EDWARD L'ESTRANGE*, Anatomist to the Royal College of Surgeons, Ireland; 31, Harcourt street, Dublin.
- 1883 *LEESON, JOHN RUDD*, M.D., C.M., Clifden House, Twickenham.
- 1869 *LEGG, JOHN WICKHAM*, M.D. C. 1886. *Referee*, 1882-5. *Lib. Com.* 1878-85. *Trans.* 2.
- \*1872 *LITTLE, DAVID*, M.D., Senior Surgeon to the Royal Eye Hospital, Manchester; Ophthalmic Surgeon to the Manchester Royal Infirmary; Lecturer on Ophthalmology at the Victoria University; 21, St. John street, Manchester.
- \*1889 *LITTLE, JAMES*, M.D., Physician to the Adelaide Hospital; Consulting Physician to the Rotunda, St. Mark's, Steevens', and the Children's Hospitals; 14, Stephen's Green North, Dublin.
- 1871 *LITTLE, LOUIS STROMEYER*, Shanghai, China.
- 1860 *LONGMORE, SIR THOMAS*, C.B., Hon. Surgeon to H.M. the Queen; Surgeon-General, Army Medical Staff (Retired); Foreign Corresponding Member, Académie de Médecine; Assoc. Soc. Chir. de Paris; Officer of Legion of Honour; The Paddock, Woolston, Hants. *Trans.* 2.
- 1894 *LOWE, THOMAS PAGAN*, 16, The Circus, Bath.
- 1883 *LUND, EDWARD*, Emeritus Professor of Surgery, Victoria University, Manchester; Consulting Surgeon to the Manchester Royal Infirmary; 22, St. John street, Manchester.
- 1867 *MABERLY, GEORGE FREDERICK*, Mailai Valley, Nelson, New Zealand.
- 1889 *MACALISTER, DONALD*, M.A., B.Sc., M.D., Physician to Addenbrooke's Hospital; Lecturer on Medicine, St. John's College; University Lecturer in Medicine; St. John's College, Cambridge.
- 1887 *MACDONALD, GEORGE CHILDS*, M.D.
- 1866 *MACGOWAN, ALEXANDER THORBURN*, M.D.

*Elected*

- \*1859 *M'INTYRE, JOHN, M.D., LL.D.,* Odiham, Hants.
- 1876 *MACKAY, EDWARD, M.D.,* Senior Physician to the Royal Alexandra Hospital for Sick Children; Assistant Physician to the Sussex County Hospital; 3, Portland place, Brighton.
- \*1854 *MACKINDER, DRAPER, M.D.,* Consulting Surgeon to the Dispensary; The Cedars, Gainsborough, Lincolnshire.
- 1891 *MANBY, ALAN REEVE, M.D.,* Surgeon Apothecary to their Royal Highnesses the Prince and Princess of Wales at Sandringham; East Rudham, Norfolk.
- 1894 *MARRIOTT, CHARLES WILLIAM, M.D.,* Aubrey House, Bath road, Reading.
- 1892 *MARTIN, CHRISTOPHER, M.B., C.M.,* 35, George road, Edgbaston, Birmingham.
- 1883 *MAUDSLEY, HENRY CARR, M.D.,* 22, Collins street, Melbourne, Victoria.
- 1839 *MEADE, RICHARD HENRY,* Consulting Surgeon to the Bradford Infirmary; Bradford, Yorkshire. *Trans. 1.*
- 1874 *MERRIMAN, JOHN J.,* 13, The Steyne, Worthing.
- 1895 *MILLS-ROBERTS, ROBERT HERBERT,* Hafod-ty, Llanberis, North Wales.
- 1887 *MIVART, FREDERICK ST. GEORGE, M.D.,* Beaumont Lodge, Worplesdon, Wimbledon.
- 1894 *MOONEY, JOSEPH JOHN,* 35, Stretford road, Manchester.
- 1891 *MORRIS, GRAHAM,* Wallington, Surrey.
- 1894 *MORSE, THOMAS HERBERT,* 10, Upper Surrey street, Norwich. *Trans. 1.*
- 1873 *MURRAY, J. IVOR, M.D.,* 24, Huntriss row, Scarborough.
- 1881 *NALL, SAMUEL, M.B.,* Dryhurst Lodge, Disley, Stockport.
- 1889 *NAPIER, FRANCIS HORATIO, M.B.,* Cape Town.
- 1870 *NEILD, JAMES EDWARD, M.D.,* Lecturer on Forensic Medicine and Psychological Medicine in the University of Melbourne; 21, Spring street, Melbourne, Victoria.
- 1868 *NICHOLLS, JAMES, M.D.,* Trenarren, Newquay, Cornwall.

*Elected*

- 1849 *NORMAN, HENRY BURFORD*, The Manor-house, Drayton, Taunton, Somerset. *Lib. Com.* 1857.
- \*1847 *NOUSE, WILLIAM EDWARD CHARLES*, Bouverie House, Exeter.
- 1884 *OAKES, ARTHUR*, M.D., Warialda, Portarlington road, Bournemouth.
- 1880 *O'CONNOR, BERNARD*, A.B., M.D., Physician to the North London Hospital for Consumption; 25, Hamilton road, Ealing.
- 1847 *O'CONNOR, THOMAS*, March, Cambridgeshire.
- \*1855 *OGLE, WILLIAM*, M.A., M.D., late Physician to the Derbyshire Infirmary; The Elms, Duffield road, Derby.
- 1870 *OLDHAM, CHARLES FREDERIC*, India [Agents: Messrs. Grindlay and Co., 55, Parliament street].
- \*1883 *OLIVER, THOMAS*, M.A., M.D., Professor of Physiology, University of Durham; and Physician to the Newcastle-on-Tyne Infirmary; 7, Ellison place, Newcastle-on-Tyne. *Trans.* 1.
- \*1871 *O'NEILL, WILLIAM*, M.D., late Physician to the Lincoln Lunatic Hospital, 2, Lindum road, Lincoln.
- 1885 *ORMSBY, L. HEPENSTAL*, M.D., Lecturer on Clinical and Operative Surgery and Surgeon to the Meath Hospital and County Dublin Infirmary; Surgeon to the Children's Hospital, Dublin; 92, Merriion square west, Dublin.
- 1887 *PAGET, CHARLES EDWARD*, Medical Officer of Health for the County Borough of Salford; North Bentcliffe, Eccles, Lancashire.
- \*1858 *PALEY, WILLIAM*, M.D., Physician to the Ripon Dispensary; Yore Bank, Ripon, Yorkshire.
- 1887 *PARDINGTON, GEORGE LUCAS*, M.D., 47, Mount Pleasant road, Tunbridge Wells.
- 1885 *PARKER, RUSHTON*, M.B., B.S., Professor of Surgery, University College, Liverpool (Victoria University); Surgeon to the Liverpool Royal Infirmary; 59, Rodney street, Liverpool.

*Elected*

- 1891 *PARKIN, ALFRED*, M.S., M.D., 5, Albion street, Hull.  
*Trans.* 1.
- 1879 *PEEL, ROBERT*, 120, Collins street east, Melbourne,  
Victoria.
- 1856 *PEIRCE, RICHARD KING*, Laggan House, Maidenhead.
- \*1856 *PEMBERTON, OLIVER*, Consulting Surgeon to the Bir-  
mingham General Hospital, President and Emeritus  
Professor of Surgery at Mason's College, Birmingham;  
H.M. Coroner, Birmingham; 65, Temple row, Bir-  
mingham. *Trans.* 1.
- 1874 *PENHALL, JOHN THOMAS*, The Cedars, Broadwas-on-Teme,  
Worcester.
- \*1879 *PESIKAKA, HORNASJI DOSABHAI*, late Hon. Surgeon to  
the G. T. Hospital (Bombay); 43, Hornby road,  
Bombay.
- \*1878 *PHILIPSON, GEORGE HARE*, M.D., M.A., D.C.L.,  
Professor of Medicine in Durham University; Senior  
Physician to the Newcastle-upon-Tyne Royal Infirmary;  
7, Eldon square, Newcastle-upon-Tyne.
- 1891 *PIERCE, BEDFORD*, M.D., The Retreat, York.
- †1841 *PITMAN, SIR HENRY ALFRED*, M.D., Consulting Physician  
to St. George's Hospital; Cranbrook, Bycullah park,  
Enfield. L. 1851-3. C. 1861-2. T. 1863-8. V.P.  
1870-1. *Referee*, 1849-50. *Lib. Com.* 1847.
- 1892 *POWELL, HERBERT ANDREWS*, M.A., M.D., M.Ch., 9,  
St. Thomas's street, Winchester.
- 1857 *VON RÁNKE, HENRY*, M.D., 3, Sophienstrasse, Munich.
- 1893 *RANKIN, GUTHRIE*, M.D., 23, Jury street, Warwick.
- 1890 *RANSOM, WILLIAM BRAMWELL*, M.D., Physician to the  
Nottingham General Hospital; The Pavement, Not-  
tingham. *Trans.* 1.
- 1854 *RANSOM, WILLIAM HENRY*, M.D., F.R.S., Consulting  
Physician to the Nottingham General Hospital; The  
Pavement, Nottingham. *Trans.* 1.
- 1882 *REID, SIR JAMES*, M.D., K.C.B., Resident Physician,  
and Physician in Ordinary to H.M. the Queen,  
Windsor Castle.

*Elected*

- 1884 *REID, THOMAS WHITEHEAD*, Surgeon to the Kent and Canterbury Hospital ; St. George's House, Canterbury, Kent.
- 1881 *RICE, GEORGE*, M.B., C.M., Sutton, Surrey.
- 1889 *RIVERS, W. H. RIVERS*, M.D., St. John's College, Cambridge.
- \*1871 *ROBERTS, DAVID LLOYD*, M.D., Obstetric Physician to the Manchester Royal Infirmary ; Physician to St. Mary's Hospital, and Lecturer on Clinical Obstetrics and Gynæcology at the Owens College, Manchester ; 11, St. John street, Manchester.
- 1889 *ROBERTS, H. LESLIE*, M.B., C.M., 46, Rodney street, Liverpool.
- 1873 *ROBERTSON, WILLIAM HENRY*, M.D., Consulting Physician to the Buxton Bath Charity and Devonshire Hospital ; Buxton, Derbyshire.
- \*1888 *ROBINSON, FREDERICK WILLIAM*, M.D., C.M., Huddersfield.
- 1889 *ROBSON, ARTHUR WILLIAM MAYO*, Professor of Surgery, Yorkshire College ; Senior Surgeon, Leeds General Infirmary ; 7, Park square, Leeds. *Trans.* 3.
- 1885 *ROCKWOOD, WILLIAM GABRIEL*, M.D., Colombo, Ceylon.
- 1850 *ROPER, GEORGE*, M.D., Consulting Physician to the Eastern Division of the Royal Maternity Charity ; and to the Royal Infirmary for Children and Women, Waterloo Bridge road ; Oulton Lodge, Aylsham, Norfolk. C. 1879-80.
- 1889 *ROSS, DANIEL McCLURE*, M.D., Cedar Lodge, Littledown Road, Bournemouth.
- 1863 *ROWE, THOMAS SMITH*, M.D., Consulting Surgeon to the Royal Sea-Bathing Infirmary ; Union crescent, Margate, Kent.
- 1882 *ROY, CHARLES SMART*, M.D., F.R.S., Professor of Pathology in the University of Cambridge ; Trinity College, Cambridge.
- 1871 *RUTHERFORD, WILLIAM*, M.D., F.R.S., Professor of the Institutes of Medicine in the University of Edinburgh ; 14, Douglas crescent, Edinburgh.



*Elected*

- 1856 *SALTER, S. JAMES A.*, M.B., F.R.S., F.L.S., Basingfield, near Basingstoke, Hants. C. 1871. *Lib. Com.* 1878. *Trans.* 2.
- †1855 *SANDERSON, JOHN BURDON*, M.D., LL.D., D.C.L. Durham, D.Sc., F.R.S., Regius Professor of Medicine in the University of Oxford; 64, Banbury road, Oxford. C. 1869-70. V.P. 1882. *Referee*, 1867-8, 1876-81. *Sci. Com.* 1862, 1870. *Lib. Com.* 1876-81. *Trans.* 2.
- 1867 *SANDFORD, FOLLIOTT JAMES*, M.D., Surgeon-Major, Medical Officer of Health of the Drayton Union Rural Sanitary District; Surgeon to the Market Drayton Dispensary; and Consulting Physician to the Market Drayton Cottage Hospital; Market Drayton, Shropshire.
- 1886 *SAUNDBY, ROBERT*, M.D., Physician to the General Hospital, and Consulting Physician to the Hospital for Women, and to the Eye Hospital, Birmingham; Professor of Medicine, Mason College; 83A, Edmund street, Birmingham.
- 1891 *SAUNDERS, FREDERICK WILLIAM*, M.B., B.C., Chieveley House, near Newbury, Berks.
- \*1861 *SCOTT, WILLIAM*, M.D., Senior Physician to the Huddersfield Infirmary; Waverley House, Huddersfield.
- 1840 *SHARP, WILLIAM*, M.D., F.R.S., Horton House, Rugby. *Trans.* 1.
- 1887 *SIDEBOTHAM, EDWARD JOHN*, M.B., Erlesdene, Bowdon, Cheshire.
- 1857 *SJORDET, JAMES LEWIS*, M.D., Villa Labrolles, Mentone, Alpes Maritimes, France.
- 1886 *SMITH, HOWARD LYON*, Buckland House, Buckland Newton, Dorchester.
- 1885 *SMITH, JAMES GREIG*, M.B., C.M., Professor of Surgery, University College, Bristol; Surgeon to the Bristol Royal Infirmary; 16, Victoria square, Clifton, Bristol. *Trans.* 1.

*Elected*

- 1894 SMITH, ROBERT SHINGLETON, M.D., Deepholm, Clifton Park, Clifton, Bristol.
- 1868 SOLLY, SAMUEL EDWIN, Colorado Springs, Colorado, U.S.A.
- 1891 STEVENS, CECIL ROBERT, M.B., B.S., Marwood House, Honiton, Devon.
- 1854 STEVENS, HENRY, M.D., late Inspector, Medical Department, Local Government Board, Whitehall; Durham Lodge, St. Margaret's road, Twickenham.
- 1884 STEWART, EDWARD, M.D., Brook House, East Grinstead.
- \*1879 STIRLING, EDWARD CHARLES, M.A., M.D., Senior Surgeon to the Adelaide Hospital; Lecturer on Physiology in the University of Adelaide, South Australia [care of Messrs. Elder and Co., 7, St. Helen's place].
- 1865 STOKES, SIR WILLIAM, M.D., M.C., Surgeon to the Meath Hospital; 5, Merrion square north, Dublin. *Trans.* 1.
- 1871 STRONG, HENRY JOHN, M.D., Consulting Surgeon to the Croydon General Hospital; Colonnade House, The Steyne, Worthing.
- \*1890 SYMPSON, E. MANSEL, M.A., M.D., B.C., 3, James street, Lincoln.
- 1870 TAIT, LAWSON, Surgeon to the Birmingham and Midland Hospital for Women; 7, The Crescent, Birmingham. *Trans.* 6.
- 1886 TEALE, THOMAS PRIDGIN, M.B., F.R.S., Consulting Surgeon to the Leeds General Infirmary; 38, Cookridge street, Leeds.
- 1890 THOMAS, WILLIAM ROBERT, M.D., Little Forest, Bath road, Bournemouth.
- 1891 THOMSON, JOHN ROBERTS, M.D., Monkchester, Bournemouth.
- 1881 THOMSON, WILLIAM SINCLAIR, M.D. (Travelling).
- 1883 THURSFIELD, THOMAS WILLIAM, M.D., Physician to the Warneford and South Warwickshire General Hospital; Selwood, Beauchamp square, Leamington.

*Elected*

- 1880 *TYT, WILLIAM JAMES*, 8, Lansdowne place, Clifton, Bristol.
- \*1871 *TREND, THEOPHILUS W.*, M.D., Physician to the Royal South Hants Infirmary; 1, Grosvenor square, Southampton.
- \*1881 *TREVES, WILLIAM KNIGHT*, Surgeon to the National Hospital for Scrofula; 31, Dalby square, Cliftonville, Margate.
- 1867 *TROTTER, JOHN WILLIAM*, formerly Surgeon-Major, Coldstream Guards; 4, St. Peter's terrace, York.
- 1873 *TURNER, GEORGE BROWN*, M.D., The Lodge, Hemel Hempsted, Herts.
- 1881 *TYSON, WILLIAM JOSEPH*, M.D., Medical Officer of the Folkestone Infirmary; 10, Langhorne Gardens, Folkestone.
- 1854 *WADDINGTON, EDWARD*, Hamilton, Auckland, New Zealand.
- \*1868 *WALKER, ROBERT*, Clovelly, Bideford.
- 1887 *WALLACE, EDWARD JAMES*, M.D., Holmbush, Grove road, Southsea.
- 1867 *WALLIS, GEORGE*, Consulting Surgeon to Addenbrooke's Hospital; 6, Hills road, Cambridge.
- \*1883 *WALTERS, JAMES HOPKINS*, Surgeon to the Royal Berkshire Hospital; 15, Friar street, Reading.
- 1894 *WARD-HUMPHREYS, GEORGE HERBERT*, Oriel Lodge, Cheltenham.
- 1846 *WARE, JAMES THOMAS*, Tilford House, near Farnham, Surrey.
- 1861 *WATERS, A. T. HOUGHTON*, M.D., Consulting Physician to the Royal Infirmary; 69, Bedford street, Liverpool. *Trans.* 3.
- 1840 *WEBB, WILLIAM WOODHAM*, M.D., Ault-sur-Somme, Somme, France.

*Elected*

- 1874 *WELLS, HARRY*, M.D., San Ysidro, Buenos Ayres, S. America.
- 1882 *WHARRY, CHARLES JOHN*, M.D., 14, Ewell road, Surbiton, Surrey.
- \*1881 *WHITEHEAD, WALTER*, F.R.S. Ed., Senior Surgeon to the Manchester Royal Infirmary, Manchester and Salford Lock Hospital, and Manchester and Salford Skin Hospital; Professor of Clinical Surgery, Owens College, Victoria University; 499, Oxford road, Manchester. *Trans.* 1.
- 1885 *WHITLA, WILLIAM*, M.D., Professor of Materia Medica and Therapeutics, Queen's College, Belfast; Physician to, and Lecturer in Medicine at, the Belfast Royal Hospital; Consulting Physician to the Ulster Hospital for Women and Children; 8, College square north, Belfast.
- 1852 *WIBLIN, JOHN*, The Hermitage, Clewer, Windsor. *Trans.* 1.
- \*1870 *WILKIN, JOHN F.*, M.D., M.C., The Warren, Beckenham, Kent.
- 1837 *WILKS, GEORGE AUGUSTUS FREDERICK*, M.D., Stanbury, Torquay.
- \*1883 *WILLANS, WILLIAM BLUNDELL*, Much Hadham, Herts.
- \*1859 *WILLIAMS, CHARLES*, Senior Surgeon to the Norfolk and Norwich Hospital; 48, Prince of Wales road, Norwich.
- 1887 *WILSON, ARTHUR HERVEY*, M.D., 504, Broadway, Boston, U.S.A.
- 1863 *WILSON, ROBERT JAMES*, 7, Warrior square, St. Leonard's-on-Sea, Sussex.
- 1889 *WISE, A. TUCKER*, M.D., Davos Platz, Switzerland.
- \*1850 *WISE, ROBERT STANTON*, M.D., Consulting Physician to the Southam Eye and Ear Infirmary; Beech Lawn, Banbury.
- 1883 *WOOD, WILLIAM EDWARD RAMSDEN*, M.A., M.D., The Priory, Roehampton.

*Elected*

1879 *WOODWARD, G. P. M., M.D.*, Deputy Surgeon-General ;  
157, Liverpool street, Hyde park, Sydney, New South  
Wales.

1892 *WRIGHT, ALMROTH EDWARD, M.D., Ch.B.*, Oakhurst,  
Netley, Hants.

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[Fellows are reminded that they are responsible for the correctness of the descriptions in the foregoing lists, and it is particularly requested that any change of Title, Appointment, or Residence may be communicated to the Hon. Secretaries before the 1st of July in each year.]



# ROYAL MEDICAL AND CHIRURGICAL SOCIETY

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## ANNUAL GENERAL MEETING

*Friday, March 1st, 1895, at 5 p.m.*

JONATHAN HUTCHINSON, F.R.S., President, in the Chair.

J. MITCHELL BRUCE, M.D., } Hon. Secs.  
RICKMAN J. GODLEE, M.S., }

The minutes of the last Annual General Meeting were read and confirmed.

The President nominated as scrutineers Dr. Lee Dickinson and Dr. William Hill, and requested them to superintend the ballot, at the same time declaring it open till 6 o'clock.

The President then called upon the Senior Hon. Secretary to read the

## REPORT OF THE COUNCIL.

The ordinary work of the Society has this year been carried on with success, but without any marked feature calling for special comment. In order, however, to increase the usefulness of the Society, the Council has made

the following changes in connection with the method of accepting papers, and publishing the *Transactions*.

1. Papers accepted for reading are thereby accepted for publication—either in the *Transactions* or in the *Proceedings*, as may be determined by the Council.
2. Papers will be printed before being read, and be obtainable (in proof) on application at the Society's rooms.
3. Fellows may read their own papers.
4. The *Transactions* are to be published in three parts (in paper covers) during the year, viz., on February 1st, May 1st, and August 1st, as well as annually (bound as heretofore); and Fellows are free to choose in which form they will receive them.
5. Papers are to be issued separately for sale (by the Society only) as early as possible after the issue of the part, at a price to be affixed to each.
6. Some of the ordinary meetings are to be devoted to discussions and demonstrations on special subjects, to be introduced by a short paper or papers by Fellows of the Society, or others, at the request of the Council.

The alterations in the Bye-laws rendered necessary by these changes will be submitted for confirmation.

The Council trusts that these changes will be appreciated by the Fellows, and will attract a larger number of communications on subjects of immediate interest from authors who hitherto may have hesitated to present their work to our Society in consequence of the lengthened period that has often elapsed between the reading and the publication of papers.

In June last a Special Meeting of the Society was held, to receive from Surgeon-Col. Lawrie a communication on "The Results of the Hyderabad Chloroform Commission." The Meeting was largely attended, and the discussion was of exceptional interest. The paper and a verbatim report of the discussion were published in the form of a special number of the *Proceedings*.

The labours of the House Committee in the management of the Society's affairs have been uniformly suc-



cessful, and have proved the wisdom of the change by which the Council transferred to this Committee the direct control of its business affairs.

Two tenants have left us during the year, but were immediately replaced by others, and the end of the year found the Society's fixed rent-roll considerably increased, while there has been no falling off in the occasional lettings.

The Honorary Librarians report as follows :

" We are pleased to be able to report that the past year has shown a steady increase in the usefulness and popularity of the Library. It has never before been so largely used, and never before has so large a number of books been taken out on loan.

" A new supplement to the catalogue has been issued, which contains all the books added to the Library during the year 1893, and also some donations of tracts and books which had been received during the building operations, and which it had been impossible to catalogue during that busy time.

" During the year the Resident Librarian has introduced the practice of type-writing day by day the titles, with subject cross-references, of all new books added to the Library, and these are placed conveniently for the use of Fellows. A further convenience to those readers who like as far as possible to help themselves has been provided in the shape of a ground-plan of the Library, showing clearly the location of every bookcase in the Library, and of a guide list to the location of those serial and academic publications which the exigencies of space make it necessary to shift from time to time, and of which the shelf numbers are for that reason not to be found in the catalogue.

“The subscription to Mr. Lewis’s library has been increased, so that now the Society is entitled to the use of 45 volumes of new books at a time. This enables the Resident Librarian to place on the shelves duplicates of all new books which are much in demand, and of text-books which are reserved for use in the Library only. During the year 114 volumes have been obtained from Mr. Lewis in this way; and it is clear that the plan is not only economical, but of much greater practical use to the Fellows than if the amount spent in the subscription had been spent in the absolute purchase of books.

“In order to utilise, for the benefit of others, the labours of those Fellows who from time to time search for references in the Library, the Resident Librarian asks that the notes thus prepared should be handed to him, and he undertakes to return to the compiler a fair typed copy of the notes, and to file another copy in a portfolio which has been placed in the Library for this purpose. There can be no doubt that in course of time a most valuable series of special bibliographies might be obtained for the use of Fellows in this way.

“During the year 526 new books and pamphlets have been added to the Library, exclusive of the large number of volumes of periodicals. The accounts show that a larger amount has been spent both upon the purchase of books and upon binding than usual.

S. J. GEE, M.D., } *Hon.*  
J. W. HULKE, } *Librarians.”*

The following report has been received from the Secretary of the Committee on Climatology and Balneology:

“The first volume of the Report of the Committee on Climatology and Balneology is in the press,

and will shortly be published by Messrs. Macmillan. This volume will include the Medical Climatology of the South Coast of England and the Channel Islands, and the more important Mineral Springs and Baths of England and Scotland. It is proposed that a second portion of the Report should deal with the climatology of the remainder of England and Wales, and also with the minor Spas."

The Honorary Treasurers report as follows :

"The finances of the Society are in an improving and thoroughly satisfactory condition. The receipts during the year 1894 amounted to £4140 17s. 2d., and the payments to £3983 8s. 6d., leaving a balance of £157 8s. 8d., and enabling the Society to begin the present year with cash in hand to the amount of £938 2s. 8d., as against £775 14s. last year.

"There has been a slight diminution in the annual subscriptions of Resident Fellows, but the decrease has been more than counterbalanced by an increase in the number of entrance fees, and a large increase in the subscriptions of Non-Resident Fellows.

"The Society's rental from tenants with whom it has leases now exceeds £2450 a year, and the occasional letting of its rooms brings in about £80. The total income from these sources may thus be placed at somewhat over £2500 a year, whilst the debenture interest amounts to rather less than £1400 a year, leaving a large margin, after deducting the rent of our Berners Street premises, for keeping up the buildings and paying the increased expenses entailed by our new quarters. When the Society entered on its present premises the floating debt amounted to about £2000 ; this had been reduced by the

beginning of last year to £533 14s. 10d., and since that date a still further reduction has been made.

“The Treasurers think it right to remind the Council that £100 will have to be set aside this year for the sinking fund to meet the debenture loan, but notwithstanding this charge they believe that the outstanding liabilities can be still further reduced.

“The whole of the expenses incurred on account of building, altering, and moving into our present premises have now been paid off, with the exception of the small sum of £20, which has been retained as a guarantee for the due fulfilment of a contract.

W. S. CHURCH, M.D., } *Hon.*  
J. A. BOSTOCK, } *Treasurers.”*

The Roll of the Society now contains the names of 503 Resident Fellows, 279 Non-Resident Fellows, and 17 Honorary Fellows.

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The Junior Treasurer, Dr. Church, then read the Annual Statement of Accounts.

He expressed his regret that the Senior Treasurer had been unable to attend that afternoon, because the bulk of the work was done by the Senior Treasurer. He would only call attention to what was stated in the Treasurers' Report, namely, the exceedingly satisfactory position in which the Society found itself. There was an income from tenants holding leases amounting to upwards of £2500 per annum. If they deducted £450 per annum, which in the course of twelve years they would cease to receive from their old premises in Berners Street, and if they also deducted £1400 a year for debenture interest, there still remained £600 a year to meet the increased expenditure necessary for the maintenance of their new premises and the increased cost

of service. The statement showed how wise the Council had been in their day in moving to Hanover Square, and might put to rest the doubts that he had heard expressed in that room as to whether the Society was in a position of financial stability. He would be very pleased to answer any questions, and Mr. Mundy, one of the auditors, was also present for the same purpose.

Mr. T. HOLMES said he had only one question to ask, and that was a small one. In the payment to be made to the bondholders this year, ought the sum not to be slightly larger seeing that the Society had received life subscriptions amounting to £101 17s.?

Dr. C. THEODORE WILLIAMS admitted that it was a very satisfactory balance-sheet, and it struck him that the Society had been very successful since it entered its new premises, such success being indisputably largely due to the energy and business skill of their indefatigable librarian, Mr. MacAlister. He, however, was not quite satisfied with things as they were. He would like to see a clear revenue of £2500 from Fellows' subscriptions, otherwise, although a learned Society, it looked as if they were carrying on a very large, and for the matter of that a very successful, commercial concern. They had a large house and let it very advantageously, but he would like to see the house being used exclusively by the Society. He hoped that this would one day come to pass. In respect of the other point, he heartily congratulated them upon the capital change they had made in reference to the printing of papers. He hoped it would add very largely to the prosperity and popularity of the Society. About the last rule, for instance: if that rule had been framed twenty years ago that excellent Society, the Clinical Society, would probably never have been formed. The report was most satisfactory from beginning to end, particularly in respect of the financial situation. He hoped the day would come when they would pay not only £100 but a great many hundreds. It was a very considerable debt, and it could only be when that was paid

off that they would have a larger part of the premises at their disposal.

Dr. CHURCH said he was glad Mr. Holmes had asked his question. It had not been thought necessary to allude to that point in the report. He pointed out that the bye-laws directed such sums to be paid over to the trustees of the debenture holders "on the direction of the Council," but so far the Council had not called upon them to pay over the composition fees for that particular purpose. The feeling was that until they had quite got round the corner, and got out of any arrears in respect of the expenses of moving, no such directions should be given. They had not wished to be too hopeful in their report, but he trusted that before many years they would be in a position to use their premises for themselves exclusively. In the meantime he could give them the assurance that financially they were a perfectly sound Society.

The PRESIDENT said it was not his desire to supplement in any way the very clear statement made to them by Dr. Church, but he thought he ought to say that that morning a letter had been addressed to him by the late Senior Treasurer, Dr. Hare (whom they all knew was most thoroughly conversant with every detail of the Society's finances), and who congratulated the Society on its sound financial position and expressed his conviction that every year would witness an improvement in its affairs.

The PRESIDENT then moved and Mr. GODLEE seconded—"That the Report of the Council, together with the Treasurers' audited Statement of Accounts be adopted, and printed in the next volume of the *Transactions*."

Carried unanimously.

The PRESIDENT then moved and Mr. GODLEE seconded—"That the changes in the Bye-laws recently made by the Council, viz.

That in Chapter XIII, Sect. ii, the words 'accept-

ance and ' be inserted before 'publication.' In Sect. iv the words 'acceptance and' be inserted before 'publication.' In Sect. v that the words after 'propriety' be struck out, and the words 'of accepting such papers as may be offered to the Society, and also upon the form of their publication' be substituted. In Sect. vi, that the words 'offered to' be substituted for the words 'read before,' and that the words following '*Transactions* of the Society' be struck out. That in Chapter XIV, Sect. iv, after the word 'volume' the words 'or part which contains the paper' be inserted. That in Chapter XV, Sect. i, line 9, all the words after 'Council' be struck out, and the words 'to be dealt with as may seem most advisable' substituted.

be and are hereby confirmed."

Carried unanimously.

The PRESIDENT delivered the ANNUAL ADDRESS.

The PRESIDENT then called upon the scrutineers to close the ballot and announce the result, and they presented the following report :

"The following gentlemen have been unanimously elected to the respective offices for which they have been nominated :

*President.*—Jonathan Hutchinson, F.R.S.

*Vice-Presidents.*—Charles John Hare, M.D., John Harley, M.D., J. Warrington Haward, John Langton.

*Honorary Treasurers.*—William Selby Church, M.D., John Ashton Bostock, C.B.

*Honorary Secretaries.*—John Mitchell Bruce, M.D., Robert William Parker.

*Honorary Librarians.*—Samuel Jones Gee, M.D., Rickman J. Godlee, M.S.

*Members of Council.*—Francis Charlewood Turner,

M.D., Frederick Thomas Roberts, M.D., Frederick Taylor, M.D., Felix Semon, M.D., William Ewart, M.D., John Hammond Morgan, Arthur Edward J. Barker, Reginald Harrison, Frederick Treves, Thomas FitzPatrick, M.D.

W. LEE DICKINSON, } *Scrutineers.*  
WILLIAM HILL, }

On the motion of the PRESIDENT the scrutineers were thanked for their services.

Mr. HEATH then moved, and Dr. ALTHAUS seconded—“That the best thanks of the Society be given to the President for the address just delivered; and that he be requested to allow it to be printed in the next volume of the *Transactions*.”

This was carried unanimously, and the PRESIDENT replied.

Dr. C. THEODORE WILLIAMS then moved and Dr. SAVAGE seconded—“That the best thanks of the Society be given to the retiring Vice-Presidents, Dr. Pavy and Mr. Pick, for their services during their term of office.”

This was carried unanimously.

Mr. T. HOLMES proposed—“That the most cordial thanks of the Society be given to the retiring Honorary Secretary, Mr. Godlee, for his valuable services during his term of office.”

Mr. Holmes said that he was in a position to speak of his own knowledge of the services rendered by Mr. Godlee on the House Committee. He was glad they were not going to lose his services altogether, seeing that he had been proposed to take the post rendered vacant by the death of Mr. Hulke, namely, that of Honorary Librarian. Commenting on the fact that upwards of £200 had been spent on the acquisition of books during the past year, he expressed the hope that Mr. Godlee in his new functions would follow the example of Mr. Hulke.

The PRESIDENT was glad to be able to affirm the



devotion of Mr. Godlee to his duties, and as President he was able to speak from personal knowledge. The Secretaries were *ex officio* members of all committees, and Mr. Godlee had been a most constant attendant at the meetings of the Library Committee.

Mr. R. W. PARKER seconded the resolution, and it was carried unanimously.

Mr. GODLEE replied.

Dr. SEMON then moved and Mr. KEETLEY seconded—  
“That the best thanks of the Society be given to the retiring members of Council, Dr. Coupland, Dr. Thin, Mr. Bennett, and Mr. Doran, for their services to the Society during their respective terms of office.”

This was carried unanimously.

In bringing the meeting to a close the President said he desired to offer his hearty thanks to his colleagues for the help rendered to him in the discharge of his duties, and he would like to include in this acknowledgment one who was not a member of the Council nor a Fellow, but to whom they were greatly indebted for his unflagging zeal and valuable services. He referred to the Resident Librarian, Mr. MacAlister.

## Receipts.

	£	s.	d.	£	s.	d.	£	s.	d.
To Balance on 1st January, 1894 :									
Cash in hand .. .. .					135	10	10		
,, at Bankers .. .. .					640	3	2		
								775	14 0
„ <i>Subscriptions, Fees, &amp;c.</i> :									
426 Annual Subscriptions at £3 3s. ..	1341	18	0						
26 Entrance Fees at £6 6s. ..	163	16	0						
23 Non-resident Subscriptions at £1 1s. ..	24	3	0						
7 Composition Fees (Life) ..	101	17	0						
1 Do. Fee (Trans.) ..	8	8	0						
Fine .. .. .			6						
				1640	2	6			
„ <i>Transactions and Proceedings</i> :									
Sold by Messrs. Longmans ( <i>Transactions</i> )	50	5	2						
,, Librarian ( <i>Proceedings</i> ) ..		14	2						
,, „ ( <i>Catalogues</i> ) ..		4	0						
					51	3	4		
„ <i>Rents received</i> .. .. .				2436	18	5			
„ <i>Interest</i> :									
On Permanent Endowment Fund ..					12	12	11		
								4140	17 2

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£4916 11 2

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## MARSHALL HALL

(TRUSTEES: *Walter Butler Cheadle, M.D.*,

The amount of Stock (Consols) standing to credit of this

## PERMANENT

(TRUSTEES: *Walter Butler Cheadle, M.D.*,

New South Wales 4% Inscribed Stock

J. A. BOSTOCK, }  
 W. S. CHURCH, M.D., } *Treasurers.*

## Payments.

				£	s.	d.	£	s.	d.
By Rent, Rates, and Taxes .. ..	..	..	..	..	..	..	238	9	9
" Lighting, Heating, and Cleaning .. ..	..	..	..	..	..	..	269	9	6
" Repairs, Furniture, &c. .. ..	..	..	..	..	..	..	93	18	6
" Meeting Expenses .. ..	..	..	..	..	..	..	22	19	6
" Printing, Stationery, and Stamped Envelopes .. ..	..	..	..	168	11	6			
Stamps (other than the above) .. ..	..	..	..	7	5	11			
							165	17	5
.. Officers and Servants :									
Salaries and Wages .. ..	..	..	..	..	..	..	806	16	8
" Library :—Books and Binding .. ..	..	..	..	..	..	..	423	15	8
" 'Transactions' and 'Proceedings' .. ..	..	..	..	..	..	..	257	16	6
" Debentures, Interest on .. ..	..	..	..	..	..	..	1395	0	0
" Auditors' Fees .. ..	..	..	..	..	..	..	10	10	0
" Bank Charges and Cheques .. ..	..	..	..	..	..	..	5	13	4
" Miscellaneous Payments .. ..	..	..	..	..	..	..	79	3	3
							3769	10	0
Former unsettled Accounts and Contracts									
Johnstone, Norman, and Co. .. ..	..	..	..	62	10	0			
Purcell and Nobbs .. ..	..	..	..	110	0	0			
Nightingale, E. .. ..	..	..	..	41	8	6			
							213	18	6
							3983	8	6
Balances :									
Cash in hand .. ..	..	..	..	..	99	19	10		
At Bank .. ..	..	..	..	..	833	2	10		
							933	2	8
							£4916	11	2

## MEMORIAL PRIZE FUND

William Ogle, M.D., and Mr. Thomas Smith.)

Fund on the 31st December, 1894, was ... £611 16 8

## ENDOWMENT FUND

Mr. Alfred Willett, and Frederick Taylor, M.D.)

.. .. £326 7 3

Audited and approved.

WOODBURN KIRBY, MUNDY, &amp; Co.,

Chartered Accountants,

19th February, 1895.

19, Birchin Lane, E.C.

## LIST OF PAPERS

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N.B.—THE Council of the Royal Medical and Chirurgical Society deem it proper to state that the Society does not hold itself in any way responsible for the statements, reasonings, or opinions set forth in the various papers which, on grounds of general merit, are thought worthy of being published in its *Transactions*.

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ADDRESS  
OF  
JONATHAN HUTCHINSON, F.R.S.,  
PRESIDENT,  
AT THE  
ANNUAL MEETING, MARCH 1st, 1895.

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IF my distinguished predecessor in this chair were still amongst us—and would he were!—we should in all probability be now engaged in the discussion of large modifications in the organisation of our Society. It was an object upon which Sir Andrew Clark had set his heart, that the Royal Medical and Chirurgical Society should develop into an Academy of Medicine. Nor was this an ambition which had originated in his brain, or in which he stood alone. He had in some sense inherited it from those whose earlier schemes had failed; but such was his energy and zeal that when he took it up, the hope that it would be realised gained much in strength. I am old enough to remember well an amalgamation scheme which seemed to be near completion about a quarter of a century ago, and in which, if I mistake not, the same high-sounding name, “An Academy of Medicine,” was also used. It was again employed by Sir Andrew Clark’s immediate predecessor, Mr. Holmes, who, in his address on vacating this chair, spoke of “those who believe that our Society has entered on a new phase of its life, . . .

that it will develop into a grand central institute or academy of medicine, which will naturally attract and absorb into its own body many, if not all, of the minor societies." Nor during the year which we close to-day have these projects been wholly at rest, for Sir Edward Sieveking, a former President to whom the Society owes very much, addressed to your Council not long ago a letter which was intended to stimulate their zeal in this and other useful directions.

Such, then, having been the aspirations of three of my recent predecessors, it may perhaps not be unsuitable that I should venture to offer a few remarks on the present position and prospects of our Society. We have been in occupancy of our new house for five years, and it is not necessary that I should enlarge on the great advantages which it has afforded us. The accommodation in our library and reading rooms is now everything which we could desire; our staff of officials has been proportionately increased, and nothing can exceed the efficiency and zeal with which our Librarian secures the success of all that belongs to his department; our rooms for meetings and for committees are commodious, and we receive now as tenants not only a majority of the London societies devoted to special branches of medical science, but also several others which have no direct connection with the profession. Aided by these sources of income our Treasurers, as you have learnt from the Report which has been read, are able to assure us that the Society's finances are in a perfectly satisfactory condition. Mr. Holmes,—to whose judgment and devotion to the Society's interest during the critical period of our removal from Berners Street we owe more than we can acknowledge, in the address to which I have already alluded, made it his business to prove, by the citation of figures with which he was familiar, that all was then promising well. Three more years have passed, and his anticipations have been more than justified. Since then debts in connection with the removal have been cleared off, and our income



has largely increased. I am not in a position to imitate Mr. Holmes by giving you details, but on the authority of our Treasurers I may state that, notwithstanding the greatly increased expenditure, the Society's position is much more satisfactory than ever it was in our smaller home; and whilst there is no room for extravagance, there is every prospect that, with ordinary prudence, the Society will be able both in the immediate and more remote future to keep its expenditure well within its income. On this point, then, we may rest assured, and the problem before us in respect to any proposals for future developments is to take care that they shall be such as will either not incur expense, or bring with them their own reimbursement. We are not in a position, and perhaps never may be,—for no Erasmus Wilson has ever befriended us with a legacy,—to launch out into new spheres of usefulness which involve expense. Should a stroke of good fortune such as I have hinted at ever come to us, the Council would know well what to do with its wealth, for our library, although excellent, is as yet far from being complete; but in the meantime we must only look to the ordinary and assured sources of income.

Now as to proposed amalgamation of the London societies, and the constitution of an Academy of Medicine, I may perhaps be permitted to say that personally my ambition for our Society does not soar so high. I was at the time very familiar with the details of the scheme which was developed some years ago, and my conviction then was, and still is, that neither the Smaller Societies nor the Medical and Chirurgical itself would be gainers by such changes. The affairs of the Pathological, the Clinical, and the Obstetrical Societies are managed better by their own councils with their own responsible officers than they would be if they were merely branches of a larger body. More zeal accrues to each, and better work and more of it is done on their present basis than would be if amalgamated. So far as accommodation under the same roof goes, the amalgamation is already effected, and in reference to the

expense of publishing Transactions it is very doubtful whether anything could be gained. As regards the constitution of an Academy of Medicine, which should have other functions than those which our Society now exercises, it may, I think, be doubted whether such aspirations are wise. That the Royal Medical and Chirurgical Society should seek to become a power in the state is probably not to be desired. Let us leave all that concerns medical education and medico-legal matters to the two Royal Colleges, the Medical Council, the British Medical Association, and the medical press. Should those in Government authority see fit to consult us, we shall, no doubt, be found equal to the occasion ; but let us not be tempted to seek the position of advisers, or feel in the least humiliated if we are passed by. Our vocation is the improvement of medical knowledge, and to that let us be true.

If, however, we put aside these more comprehensive proposals, it by no means follows that we ignore all suggestions of modification and development. It may be that there are directions on a somewhat lower plane in which much may with advantage be done. Your Council has during the past session directed its attention to the smaller number of papers which are, year by year, offered for its acceptance, and the diminishing interest which appears to be taken in its meetings. The causes of these occasions for regret are not far to seek. In the early days of the Society it enjoyed nearly a monopoly as an arena for the discussion of first-class papers on medical subjects. Its 'Transactions' offered almost the only place of publication in which such papers were certain of permanent recognition. It is not so now ; the press has developed, and on all hands new societies, each publishing its own Transactions, have sprung up, several of which, inasmuch as they cultivate specialties and offer a specialist audience, afford advantages superior to our own. There is nothing in this to be regretted, for the stream of medical science is flowing with far fuller stream and greater force than in the former days. The river of knowledge for the benefit of

man is far from suffering any loss, but there is in our case some risk that one of its ancient tributaries may be drained by others and left to dwindle. Under such circumstances what is to be done? Clearly we must adapt ourselves to the changing conditions of the time, and make it our business to find out improved methods of work. It is, I think I may venture to say, under impressions somewhat of this kind that your Council has during the past session adopted several modifications in reference to the conduct of our meetings. Henceforth the acceptance of a paper for reading is to be acceptance for publication also, and thus an impediment to the putting the paper in type beforehand is removed. All papers are to be put in type beforehand, and will be at the disposal of our Fellows a few days before the meeting is held at which they will be read and discussed. I may suggest that it is possible under this plan that papers may but seldom be read in full, the author being requested rather to explain his salient points, and thus save the time of the meeting and the patience of the audience. In this way much more time will be secured for the discussion, and those who take part in it will have had better opportunity for preparation. Surely it is in the discussion that the principal advantage of a Society's meeting over mere publication consists. Speaking for myself, I shall not be at all surprised if at no distant time it should become usual to publish the paper in the journals a week before the discussion, and thus permit all interested to come to the meeting with minds well prepared. Such a plan seems to me perfectly reasonable, and not unlikely to give us larger meetings and better debates. Another proposal which the Council has adopted is that of having from time to time, and perhaps not very infrequently, pre-arranged discussions on topics of interest. Here, again, we have an expression of belief that in these days the value of a meeting consists far less in listening to a long disquisition, whether lecture or paper, by one man, than in the opportunity afforded for obtaining expressions of experience from many observers. By no means is it a

primary object to encourage the debating spirit or to afford opportunities for mere verbal display. Rather it is hoped that these discussion evenings will be enriched by the production of patients and specimens, and distinguished by the accumulation of new facts quite as much as by the expressions of opinion and criticism. It is believed there are many subjects very suitable for such discussion, respecting which no single individual would deem that he possessed original matter sufficient to warrant him in offering a paper. We require from those who send us papers that they shall give us original facts or novel views, but upon those who at the request of the Council may undertake to open discussions no such burden will be laid. It will be sufficient that a clear exposition is afforded as to the present state of knowledge, and the lines traced by which further research should be guided. It is thought probable that in selecting subjects for these discussions the Council may be able to secure that topics of great general interest may be brought before the Society, which might never have come up in the form of original papers.

Other proposals, having for their object increase of interest in our meetings and increase in the value of our 'Transactions,' have been mooted, but as they have not as yet in any sense received the sanction of the Council it is perhaps premature for me to refer to them. Although our title is the Royal Medical and Chirurgical Society of London, we have never been exclusively metropolitan. Our list of Fellows has always been graced by many Provincial and even some Colonial names, and our 'Transactions' have often been enriched by their contributions. It may be that, in the future, means may be found for bringing into closer association with ourselves the members of provincial, colonial, and even foreign societies having similar objects. Amongst the vast modifications which social life has experienced during the present century, unquestionably the increased facilities for moving from place to place are the chief. It is now by no means difficult for provincial residents to attend London meetings,

and even from far more distant places the representatives of medicine and surgery may often find their way to our metropolis, or we to theirs. The Royal Medical and Chirurgical Society should have no rivals, but it should make many friends. If it should be found practicable to devise some affiliation scheme which should enable the members of sister institutions to have access to our rooms when visiting London, and to attend our meetings on equal footing with ourselves, I feel sure that much mutual advantage would accrue. It is perhaps possible also that something might be done by central publication of reports to diminish the loss of valuable clinical work which now results from isolated and often too ephemeral record. All this is, however, for the present a mere suggestion, and again I must say that I speak for myself only, and not by authority.

A kindly and commendable custom has imposed upon your President the duty of reminding you in his Annual Address of those Fellows and friends whom we have lost during the year. The practice, now so general, of giving in the journals of the day full, and usually very accurate, memoirs of deceased members of our profession has rendered this duty less binding than it once was. It is one, however, which we may trust will never be wholly neglected.

On the present occasion I am glad to say that I have to mention only few names, but the list, though short, includes some heavy losses. From our list of Honorary Fellows two names must be taken.

Professor *Adolphe Hannover*, of Copenhagen, who was elected nearly thirty years ago, died last July. It is more than half a century since his 'Microscopic Researches on the Nervous System' gained him reputation, and having regard to the rapid progress made in this department of anatomy, it is not surprising, nor in the least disparaging, that his fame should have somewhat waned. Not long after this work he was engaged in important investigations

as to the excretion of carbonic acid, and wrote also on the 'Anatomy and Physiology of the Eye.'

*Professor Hermann Ludwig von Helmholtz* died in Berlin last September. He was in his seventy-second year, and had been on our Fellows' list since 1873, the same year in which he received from the Royal Society the distinction of the Copley Medal. He was a man of profound knowledge, originality, and of unwearied industry. His first volume on the 'Conservation of Force' was published when he was only twenty-six, and from that time he continued to labour most successfully and in varied directions for the advancement of physical and physiological science. His discovery of the means by which to inspect the fundus of the eye and his invention of the ophthalmoscope was probably to him, and in relation to his other labours, a very small matter.

Of our ordinary Fellows six have died during the year. One of these, *Mr. Barnard Holt*, came near to the honour of being our senior, for his name had been on our list close upon fifty years. He was elected in 1846, and died in March, 1894. In the earlier and middle parts of his career *Mr. Holt* had enjoyed a good practice, and was in much repute as an ingenious and practical surgeon. In later life he allowed his love of horses to absorb much of his time and attention. He was a man of genial manners and a kind heart. He never got old, but to the last preserved a florid and almost youthful complexion. To within a very short period of his death it was no unusual thing to meet him on the box of a brake, displaying his skill in the management of a pair of restive horses.

A miserable street accident, one dark evening in October last, deprived the profession of a physician who was second to none amongst us in his attainments or in his charm of character. *Dr. Octavius Sturges* was born in London in 1833, and died at the age of sixty-one, whilst still in vigorous health and full work. He was at the time of his death senior censor to the College of Physicians, and senior physician to the Westminster Hospital and to the

Hospital for Sick Children. His two works on Pneumonia and on Chorea are classical.

The short time at my disposal compels me to pass by the names of *Dr. A. G. Medwin*, *Dr. A. E. Butler*, and *Dr. C. Cyril Hicks*, without venturing to offer any biographical details. The two latter were comparatively young men, but Dr. Medwin had been thirty years on our roll. They were all men who had done good service in the cause of our profession, and whose lives had been alike honourable to themselves and useful to others.

Of *Mr. Hulke* it might perhaps be sufficient to write that his much-lamented death left vacant the Foreign Secretaryship to the Geological Society, the Senior Surgeoncy to the Middlesex Hospital, the office of Honorary Librarian in our own Society, the Presidency of the Clinical Society, and that also of the Royal College of Surgeons. That it was possible for one man's death to vacate all these various offices is a sufficient indication of his varied powers, and of the estimation in which he was held by his colleagues. There were, however, in addition to what I have named a host of minor but yet important posts in committees and in boards of management for which successors to Mr. Hulke will have to be sought. His services in these posts were always highly valued, for he was a man of clear judgment and most punctual in his attention to all public duties. In our own Society he had been member of Council, Secretary, member of Library Committee, and lastly, up to the time of his death Honorary Librarian. No fewer than nine papers from his pen have found a place in our 'Transactions,' and he used frequently to mention for the encouragement of younger men that the first which he offered was refused. Mr. Hulke had been President of the Pathological and Ophthalmological Societies, in connection with both of which he had done much excellent work. In early life he secured the good opinion of such men as the late Dr. Robert Bentley Todd and Sir William Bowman, with both of whom he was, I believe, associated in anatomical and

physiological investigations. The Fellowship of the Royal Society was conferred upon him at the age of thirty-six, equally perhaps in connection with his successful devotion to such different subjects as histology and palæontology. At this period his days were given to the hospital, his evenings to the microscope, and his holidays to the field—geology. His discoveries in the latter were of great value, and have received the hearty acknowledgment of those best qualified to estimate their worth. He was President of the Pathological and the Geological Societies at one and the same time, and discharged the duties of the two very dissimilar posts with equal credit to himself.

Of Mr. Hulke's character and social habits it is not needful that I should speak, for we all knew him. Few, however, had probably known him longer than myself, or been associated with him as colleague in more various capacities. Our friendship dates back from the time of his tutorship at King's College, and extends through the whole of the period that we were colleagues at Moorfields, at the College of Surgeons, and elsewhere. I will not profess that we always worked shoulder to shoulder, for in truth we often differed; but of this I feel sure, that I do but register the conviction of all who knew him when I assert that I never associated with a man more upright in his conduct, or more conscientiously determined to follow what he believed to be his duty.

Thus, then, gentlemen, although our year's death-roll is not a long one, it comprises the names of three men than whom no better representatives of the highest aspects of professional life could well be found. Let Helmholtz stand for pure science, Octavius Sturges for sound clinical medicine, and John Whitaker Hulke for scientific surgery. These three men have during the year which has just past ceased from their labours, and left us to mourn their loss. They have left us also the example of their lives and the warning of their deaths. By the expression "warning" I mean only this: that we should lay to heart the uncertainties with which we are environed,



and let them serve to us as cogent motives to the prompt performance of all such professional duties as have fallen to our share. Let our lives be without hurry, but at the same time without loitering. The street cab, the chill night's summons to a patient's bedside, these and a hundred other risks lie before us, and may, with little or no notice, cut short an unfinished career. Let us, then, make our wills in time,—in other words, let us take steps that such wealth of knowledge as we have gained, during our lives of labour, be not lost unto our heirs. For, between the wealth represented by land and gold, and this far more precious kind, there is the great difference that we may easily carry it with us to the grave unless due precautions be taken. No one of the three whom I have just named rests under any imputation of negligence in this matter; they had one and all carefully and conscientiously given substance to their otherwise evanescent intellectual wealth. They had made their wills, and posterity will realise their generous bequests. In this as well as in their unflinching devotion to the pursuit of truth, and their untiring industry, their lives are examples which we shall always contemplate with pleasure and profit, reminding us, as they ever will, that—

“Spite of cormorant devouring Time,  
The endeavour of this present breath may buy  
That honour which shall bate his scythe's keen edge,  
And make us heirs of all eternity.”



**THE**  
**SURGICAL TREATMENT OF DIFFUSE**  
**SEPTIC PERITONITIS ;**  
**WITH SUCCESSFUL CASES.**

BY

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WITHOUT doubt the term diffuse septic peritonitis comprises many kinds of disease. Surgeons seem to have included in it cases in which the peritoneum was hardly altered after death; others in which its vessels were full of blood and its surface smeared with oil-like lymph; others in which there was ashen-grey lymph, with slight adhesions; others in which purulent fluid predominated, and so forth. There seems, however, a consensus of opinion that in diffuse septic peritonitis the inflammatory action has no definite limits, runs an acute and rapid course, and is almost invariably fatal.

If this is correct, it is necessary at the outset of this communication to say what kind of peritonitis is implied by the term diffuse septic peritonitis.

Fully conscious of the difficulties of definitions, I would say that in the diffuse septic peritonitis which

## 2 SURGICAL TREATMENT OF DIFFUSE SEPTIC PERITONITIS.

I refer to the inflammation is acute and has no definite limits; the serous membrane is smeared with lymph, which is either transparent and oil-like or collected in ashen-grey fibrinous flakes; adhesions are absent, or very slight; the peritoneal cavity contains a variable quantity of thin purulent fluid, which often has a fæcal odour; or it may be full of gas; the intestines are distended and paralysed; and the disease runs a rapid and fatal course when left alone.

Although the cases which come within this definition are numerous, surgical literature does not contain many instances of recovery after operative intervention.

Senn,<sup>1</sup> who has paid great attention to this subject, has recently said that diffuse septic and suppurative peritonitis are seldom, if ever, cured by laparotomy.

Writing in 1892, Körte<sup>2</sup> said that cases of general septic peritonitis with little exudation and with paralysis of the intestinal walls were not capable of improvement by direct surgical interference. Fränkel is almost as despondent.<sup>3</sup>

Mr. Treves,<sup>4</sup> in his Lettsomian Lectures, in which he exhaustively deals with the literature of peritonitis, says, "The surgical treatment of acute diffused peritonitis as a purely inflammatory affection has not been very successful, and has, indeed, been comparatively unsuccessful. This treatment consists of incision and the evacuation of the effusion. . . . It is comparatively useless."

Mr. Tait has said that when once peritonitis is completely established it is a practically incurable disease, and almost uniformly fatal.<sup>5</sup>

With the exception of the cases which I am about to narrate there has not been, so far as I am aware, a case

<sup>1</sup> 'Principles of Surgery,' Philadelphia and London, 1890, p. 301.

<sup>2</sup> 'Berlin. klin. Wochenschrift,' No. 31, 1892.

<sup>3</sup> 'Deutsche medicinische Wochenschrift,' January 22nd, 1891, p. 140.

<sup>4</sup> "Peritonitis," 'The Lettsomian Lectures for 1894,' London, p. 18; also 'Trans. of the Med. Soc. of Lond.,' 1894, vol. xvii.

<sup>5</sup> Quoted by Treves, loc. cit., p. 101.

of recovery after laparotomy for diffuse septic peritonitis in St. Bartholomew's Hospital or in the Great Northern Hospital.

Scattered throughout surgical literature, however, cases are to be found in which diffuse septic peritonitis got well after operation. Some are mentioned in Mr. Treves' lectures,<sup>1</sup> some in the 'Transactions' of the Clinical Society, and elsewhere; but they all differ from the cases which I am about to relate in not having been treated upon a definite and systematic plan.

So far as I can judge, diffuse septic peritonitis caused by perforations of the vermiform appendix have hitherto given the best results; then those in which the cause was unknown; next, those in which there was a perforation of the small intestine; and lastly, those in which the stomach or duodenum was perforated. Of course I refer to cases in which the peritonitis was fully established, and not to cases of recent perforation or laceration.

Many of the cases described under the headings of *diffuse septic peritonitis*, *general septic peritonitis*, and of *suppurative peritonitis* are probably examples of localised septic peritonitis. Moreover, as I have just said, none of them seems to have been treated upon a definite plan such as is advocated in this communication.

Like other surgeons, I have had a most disheartening experience of this class of cases. Clinically they are like acute intestinal obstruction due to mechanical causes. Therefore I have in many instances performed laparotomy, more because I was afraid of overlooking a mechanical obstruction than in the hope of curing the peritonitis.

Last year, however, I operated upon a case which was encouraging, and afforded food for reflection. A boy eight years old came into the Great Northern Hospital after having had, for at least a week, an acute septic peritonitis around the vermiform appendix. About the time of his admission there is no doubt that the peri-

<sup>1</sup> Loc. cit., p. 113, *et seq.*

tonitis became diffuse, and at the operation a diffuse septic peritonitis was found. A large quantity of purulent and feculent fluid was washed out of the pelvis, from the flanks, and from amongst the coils of intestines, which were very slightly adherent. The vermiform appendix was perforated in six places, and was removed. The abdomen was washed out and drained. Before this the boy was evidently dying; he had had absolute obstruction for five days, with frequent fæcal vomiting. His face was pale, with hollow cheeks and sunken eyes. His temperature was subnormal. Afterwards he revived with heat and stimulants, and improved so much that it seemed possible for him to recover. But two days after the operation the abdominal distension returned. He became sick again, and died towards the middle of the third day.

Before his death an endeavour was made by Mr. Kendall, the house-surgeon, to relieve the distension by thrusting a small trocar and cannula through the abdominal walls into the intestines. This was quite inefficacious.

In septic peritonitis, Mr. Malcolm<sup>1</sup> has said, "there are signs of a triple pathological state,—of peritonitis, of blood-poisoning, and of paralytic obstruction of the bowels." Now it seemed clear that of these the intestinal paralysis and distension were the fatal elements in this case. I have regretted ever since that a bolder course was not pursued, and that I did not again open the abdomen and empty the intestines.

The importance of this intestinal distension and paralysis was pointed out by Benjamin Travers, and has of recent years been better appreciated by surgeons. Senn<sup>2</sup> and Greig Smith<sup>3</sup> have drawn especial attention to it, and in preparing this paper I am indebted to their writings.

I have, however, been unable to ascertain that either

<sup>1</sup> 'Med.-Chir. Trans.,' vol. lxxi, 1888, p. 58.

<sup>2</sup> 'Intestinal Surgery,' Chicago, 1889.

<sup>3</sup> 'Abdominal Surgery,' 1888; also communication before Royal Medical and Chirurgical Society, March 8th, 1892.

of those surgeons has published a successful operation for diffuse septic peritonitis.

The distension and intestinal paralysis are not the only factors to be considered in these cases. There is in addition the septic poisoning, and owing to this and want of food, constant vomiting, pain, sleeplessness, and mental distress, the patients are in an exhausted and critical condition. Thus some of the causes of death from diffuse septic peritonitis cannot yet be overcome—the septicæmia, for instance; but the sapræmia may be cured by copious irrigation and drainage; the intestinal distension by the evacuation of gas and fæces; and the exhaustion and collapse by the methodical use of warmth and stimulants.

We now possess many means by which the exhaustion and collapse may be met. I myself have only gradually learnt the proper value and absolute necessity of the measures which I am about to advocate as a routine practice in these cases. They are as follows. Before operating the patient's condition is temporarily improved by the subcutaneous injection of strychnine (about  $\frac{1}{10}$  grain), and by enemata of brandy and hot water. These may be repeated during the operation, with the subcutaneous administrations of ether in addition. Next the operation room is heated to 70° or 75° F., and draughts excluded. A hot-water bed for the patient to lie upon during the operation is absolutely essential, and may be supplemented with hot-water bottles in the axillæ and to the feet. It is wise to wrap the limbs in cotton wool. All towels, sponges, instruments, and lotions should be hot. I have found 110° to 112° F. a suitable temperature. The anæsthetic should be used sparingly, and only pushed to prevent violent contractions of the abdominal muscles. I am inclined to think the A. C. E. mixture causes least depression. Every manipulation is carried out as speedily as possible. Slow operating means failure. The abdomen is opened by a long incision in the linea alba below the umbilicus without looking for muscular inter-

spaces. If no altered intestinal coils are quickly found, the cæcal region is at once explored. Most of the causes of mechanical or of inflammatory obstruction are to be found there. If none are found and the cæcum is distended, it is clear that the obstruction is in the large intestine, or is inflammatory. To settle this point the sigmoid flexure is next examined. If it be distended, and if there be no obstruction in the rectum, a point previously ascertained, the case is clearly not one of mechanical obstruction. This search is made easier by a sufficient extrusion of small intestines. Towels which have been sterilised and wrung out in hot water are used to keep the extruded intestines moist and warm. This is a point of extreme importance. Pursuing the search, the small intestines are more fully examined, and then the pelvic viscera. In some cases the incision must be carried upwards, or a new one made, and the stomach and duodenum explored.

If the cause of the peritonitis be met with, it is dealt with as seems best at the time. If a perforation of the small intestine is found, as in one of my own cases, it is not closed at once, but is used to facilitate the next step in the operation, namely, the evacuation of the gaseous and fæcal contents of the intestines. The gas is let out of each coil by puncturing it with a trocar and cannula about 1·5 mm. in diameter. This is introduced as often as is necessary to empty each coil. The fæcal contents are evacuated through incisions half to three quarters of an inch long into one or more of the fullest loops. This is made longitudinally, as far as possible from the mesentery. In one of my cases this incision merely enlarged the perforation; in another it enlarged a puncture which seemed disposed to leak. A little squeezing helps the gas and fæces to escape. As the coils are emptied they are washed and put back, but the incised one is kept extruded until the last. When nothing more escapes from it the opening is closed with Lembert's suture, and it is dropped back into the abdomen.



The abdomen is washed out in a definite order; first the flanks and intestines, next the iliac fossæ, and then the pelvis. A large glass drainage-tube is introduced into Douglas's pouch, and the abdomen is closed in the usual way with silkworm-gut sutures. No time is occupied with sponging to remove fluid. What remains is sucked from the drainage-tube. After the operation a skilled person is told off to combat shock with subcutaneous injections of brandy, ether, and strychnine. Subsequently no attempt is made to introduce food by the mouth, but rectal feeding is practised as follows. The rectum is washed out with tepid water, and a rectal tube left in for twenty minutes. This usually allows flatus to escape. Then about four ounces of concentrated nutrient enema, consisting of peptonised milk, peptonised meat essence, with a little good brandy and a little opium, is administered. This enema is used every three hours during the first few days, and is gradually discontinued. Thirst is relieved by enemata of tepid water. Feeding by the mouth is begun with great caution. Small quantities of fluid peptonised food are given for some weeks.

I ought to apologise for mentioning such details as some of the foregoing; they are familiar to every surgeon. Nevertheless I believe them to be no less important than the most impressive details of the operation.

In the preparation of the skin, instruments, towels, and other materials the usual precautions are taken to obtain asepsis. The mere fact of the case being already a septic one is not a reason why more sepsis should be introduced.

I will now describe a case of diffuse septic peritonitis which was successfully treated after the manner which has just been briefly described.

The patient was a boy seven years old, and born of Polish Jew parents. He was admitted under the care of Dr. Hensley, for whom I was asked to operate. He

seems to have been ailing and getting thinner for three weeks before his critical illness began. On the evening of May 26th, 1894, he began to vomit, and his abdomen became distended. He had a very slight action of the bowels on May 26th, upon which day he was admitted, but the last natural action of the bowels had occurred, it is said, five days previously—on May 21st.

When I saw the boy upon the afternoon of May 26th he was suffering from acute intestinal obstruction, with the usual distension of the abdomen, vomiting, and inability to pass flatus or fæces. The abdomen was tender all over, and exceedingly tense, tympanitic, distended, and motionless intestinal coils could be plainly discerned. The general condition was precarious. The temperature was  $100.8^{\circ}$  F., the pulse 110 and very small, and the respirations twenty-two per minute. The face was pale with hollow cheeks and sunken eyes. There was no indication for localising the source of infection, but the vermiform appendix was suspected. Recovery was clearly out of the question, and the boy had evidently but a few hours to live.

A median incision six inches long having been made below the umbilicus, it was seen that a severe acute diffuse septic peritonitis was in progress, with purulent fluid in the flanks and pelvis, with some free gas and flakes of lymph about the intestines and mesentery. These were pulled off wherever they could be reached.

The peritonitis was of the kind in which there seems to be hardly any tendency to form adhesions. The odour of the fluid and lymph was fæcal. The mesenteric lymphatic glands were much inflamed and swollen. The purulent fluid having been rapidly washed out with hot water, I soon ascertained that the cæcum and vermiform appendix were intact, but that both the cæcum and the sigmoid flexure were distended. Thus the idea of a mechanical obstruction was put aside. Next the small intestines were extruded and examined, and in the ileum a small circular aperture was found, with intestinal con-

tents running from it. I therefore proceeded to systematically empty the intestines by puncturing every distended loop with a small trocar and cannula. As the loop with the original hole in it did not empty easily the aperture in it was enlarged with the scissors, and a quantity of fæcal contents evacuated. Afterwards this opening was closed with Lembert's sutures, the intestines returned, the abdomen thoroughly washed out with hot water, and drained with the usual glass tube. During the operation a quantity of lymph was removed from betwixt the folds of the mesentery, and from amongst the coils of intestines.

This operation lasted more than an hour, but was not followed by very excessive shock. Strychnine, ether, brandy, enemata and warmth brought about an improvement. As he came round, and when we were about to wash out his stomach, a quantity of orange peel, orange pips, grape skins, and such like things were vomited up. He was fed by nutrient enemata, and at night his temperature had risen to  $101.9^{\circ}$  F. This was its highest point. Next day it became normal, and did not afterwards fluctuate. For a time after the operation his condition was precarious. His breath had a peculiar sweet offensive odour, vomiting was troublesome, and his face was dusky. However, on May 29th he was decidedly better. The glass tube was withdrawn, and a rubber one put in its place, the vomiting ceased, and he began to be fed by the mouth. The discharge from the drainage-tube was purulent, but had no fæcal odour at any time. After this he slowly but surely mended. On June 4th he ceased taking opium, and on the 5th the nutrient enemata were discontinued, and upon the same day his bowels were opened twice. After this our only anxiety was about the wound, which had suppurated, broken down, and gaped widely. At its bottom the granulating coils of small intestine were clearly displayed.

However, cicatrisation proceeded steadily, and the wound was healed at the end of July. During the early part of August he seemed to have some difficulty in swallowing,

but this disappeared. He was discharged on August 14th because he seemed quite strong and well, and was very noisy and troublesome in the ward.

For many of the foregoing details I am indebted to Dr. Brooksbank's very clear and full notes of the case. Throughout his illness the child owed much to the care of Mr. Rudolph Smith, the house surgeon, and to the nursing he received in Matthew Ward. Before his departure his abdomen had become quite soft and natural, but was rather sensitive. Adhesions were probably absent, as was proved to be the case in a similar instance described further on in this communication (see p. 16).

As regards the cause of the perforation of the ileum in this case, I felt sure that it had been something which had been eaten. When the hole was enlarged a quantity of husks escaped, and Sister Matthew afterwards showed me the husk of an almond nut which she had found amongst the vomit.

I operated upon another case of diffuse septic peritonitis on September 8th, 1894, using similar measures.

The patient was a married woman aged forty years. She was the mother of three children, of whom the eldest was sixteen years of age, and the youngest two and a half. At the last confinement the womb was lacerated, and there was possibly some pelvic inflammation. The menstruation was normal, the last period having ceased four days before her illness began. With the exception of indigestion, and pain and flatulence after food, the general health was good. Beyond this nothing in the history pointed to the existence of a gastric ulcer or of any intestinal affection.

Her illness began at 10 a.m. on September 6th. Whilst doing her household work she was suddenly seized with an abdominal pain. This was said to have been worst in the right iliac region. She did not faint or become unconscious, but was quite prostrated, and had to take to her bed. The pain was quickly followed by severe vomiting, which ceased after the subcutaneous injection of morphia.

Her bowels also ceased to act, and neither fæces nor flatus were passed.

It is possible that her illness was aggravated on September 8th by a fire which necessitated her removal from home. She was admitted into St. Bartholomew's Hospital on September 8th. She was a stout, florid woman, whose face was dusky, with an anxious expression. The pulse beat eighty per minute, and the respirations were forty. The tongue was dry, and she complained of nausea and of pain in the abdomen. There was almost complete intestinal obstruction. After admission a little flatus was passed, but an enema only brought away a few scybala. The abdomen was distended and resonant everywhere, the liver dulness being absent. There was universal tenderness, but not more in one situation than in another. Pain could not be localised. Although the abdominal walls were fat, the distended and motionless coils of the small intestines could be seen. A rectal examination revealed nothing; the os uteri was patulous, but there seems to have been no endometritis.

Dr. Norman Moore, who saw her in consultation, concurred with me that this patient was dying of acute septic peritonitis. He also agreed that although its source was unknown, a perforating gastric ulcer might be excluded. It was determined to perform laparotomy with the view of treating the peritonitis, and, if possible, of removing its cause.

Reckoning from the sudden onset of pain to the time of operation, the peritonitis was of fifty-two hours' duration. The condition which was found was quite compatible with this calculation. The patient was placed upon the hot-water bed and her limbs enveloped in cotton wool. An incision in the middle line below the umbilicus gave exit to a quantity of gas and purulent fluid, and a quantity of the latter was rapidly washed out with hot water (temp. 110° F.) discharged from an irrigator. It had no particular odour. The parietal and visceral peritoneum were acutely inflamed and covered with lymph.

## 12 SURGICAL TREATMENT OF DIFFUSE SEPTIC PERITONITIS.

This was either of the delicate transparent kind, or, in places, fibrinous and opaque. The inflammation was diffuse and unaccompanied with adhesions. All the intestines were tightly distended. The cæcum and vermiform appendix were inflamed but not perforated. The sigmoid flexure was distended. The small intestines were intact. They had to be extruded for examination, and were covered with sterilised towels wrung out in hot water. The right ovary and Fallopian tube were not seen, but they, together with the uterus, felt natural. There were more purulent fluid and flakes of lymph about the left Fallopian tube than elsewhere, and it looked very red, swollen, and œdematous. It seemed to me to be the probable starting-point of the peritonitis. The distended small intestines were now emptied of gas with a small trocar and cannula. About a dozen punctures sufficed. One continued to leak, so it was enlarged and used to empty out a quantity of bilious fæcal matter. This opened loop was kept extruded until the last, and the opening was finally closed with Lembert's sutures and the intestine was returned. Not the slightest difficulty was experienced in putting the intestines back. The abdomen was then methodically irrigated with about two gallons of water, temp. 110° F., drained with a glass tube in the pelvis, and closed and dressed in the usual way.

The patient was under the anæsthetic an hour and a quarter. Owing to the tightness and thickness of the abdominal walls the manipulations were very difficult. I gained great help by retracting the lips of the wound with a stout silk suture on either side. They were carried through the whole thickness of the abdominal wall. They are much better than retractors, and give complete command of the wound without getting into the way. I am indebted to Mr. Sheild for this device. Before closing the wound these sutures are of course removed. The operation was well borne, and the patient revived with warmth and brandy. She vomited twice during the night, at 11 p.m. and 1.45 a.m. After this the vomiting ceased and never

recurred. She was fed every three hours with concentrated, peptonised, nutrient enemata. On September 9th flatus was passed spontaneously, and there was no abdominal pain or distension. On September 11th a small quantity of chicken broth and champagne were given by the mouth, and afterwards these were gradually increased until a solid diet was reached; simultaneously the rectal feeding was gradually discontinued. The bowels acted spontaneously on September 16th, and afterwards gave no trouble. On September 11th, and for some days afterwards, the urine contained albumen. Subsequently it contained some mucus in addition, due to a slight cystitis. A catheter was passed once with proper antiseptic precautions. The wound, as usual in these cases, suppurated and gaped slightly, but healed rapidly after it had begun to granulate. The glass drainage-tube evacuated a quantity of purulent fluid, neutral in reaction, of a greenish colour, and devoid of odour. On September 14th it was changed for a rubber tube, which was gradually shortened, and finally discontinued on September 20th. The temperature was never high. Before the operation it was 100° F., and afterwards fell slightly, to rise again to 100·8° F. on the third day. It remained at this level until the tube was removed on September 20th, when it rose to 101° F., and remained thus until September 27th, when it fell abruptly from 102° F. to normal, and continued normal for a week. During her convalescence it fluctuated without apparent cause. She left the hospital on December 10th in excellent health.

During her severe illness this patient's chief trouble was sleeplessness, accompanied with slight delirium at night.

I am indebted to the clear and exhaustive notes of Mr. Giles for most of these particulars. Mr. Maidlow, the house surgeon, bestowed infinite pains upon the case, and much was owing to Sister President.

These two cases were treated upon exactly the same

#### 14 SURGICAL TREATMENT OF DIFFUSE SEPTIC PERITONITIS.

lines, and may be commented upon together. In the case of the boy the cause of the peritonitis was found and easily dealt with. In the case of the woman it remains rather doubtful. The presence of free gas in the peritoneal sac pointed more to a perforation than to infection from the tube. In cases recorded by others, and which recovered, the source of the peritonitis was not discovered.<sup>1</sup> One is rather reluctant to fall back upon a phrase, and call this a case of idiopathic peritonitis.

I do not propose to dwell any more upon all the methods which were employed, except to refer to the puncturing of the intestines. I believe that this is quite harmless when it is done with a fine trocar and cannula, and with the intestine in sight. The cannula is intended to evacuate gas, and therefore a very small one is required. A cannula suitable for the evacuation of the other intestinal contents would have to be very large, because, as in the case of the boy, they often contain bodies of considerable size. Such a cannula would be almost certain to make a hole which leaked. If the puncture made by the small trocar and cannula were to leak, it is easily closed with a suture; or, as the leak implies considerable tension, it may be converted into the opening for the evacuation of the faecal contents. This was done in my second case. There is clearly a great difference between the insertion of a trocar and cannula into coils of intestines which are in sight, and plunging them through the abdominal wall into viscera which cannot be seen. In the first the exact spot can be chosen, and a leak can be secured; in the second, large vessels may be wounded, or a fatal leakage remain undiscovered.

I first employed an incision for the emptying of intestines in diffuse septic peritonitis in July, 1888.<sup>2</sup> Since then I have seen it used in other cases, and have never known it cause faecal extravasation. It seems to be a safe and convenient procedure.

<sup>1</sup> Southam and Kelynaek, 'Medical Chronicle,' Manchester, vol. xvi, 1892.

<sup>2</sup> 'Trans. of the Med. Soc. of Lond.,' vol. xv, p. 91.



Whether the incision ought always to be closed, or whether it ought sometimes to be converted into a fæcal fistula, will require experience to determine. The immediate closure of the incision is the ideal procedure, as it obviates a second dangerous operation for the closure of a fæcal fistula.<sup>1</sup>

Hot water from the tap seems to be suitable for washing out the abdomen. It would be dangerous to use antiseptics strong enough to disinfect, and chemicals are known to be harmful to the peritoneal endothelium. It is probable, as Delbet<sup>2</sup> showed, that little is absorbed from the peritoneum for some time after profuse irrigation. I used some gallons of water at a temperature of 110° F.<sup>3</sup> in the receptacle of the irrigator.

Although in these septic cases the wound seems always to break down and suppurate, yet it seems best to close it, except the part for the drainage-tube. The sutures do not yield for some days, so that for a time they obviate the danger of intestinal prolapse during coughing or vomiting.

The foregoing cases illustrate the value of immediate puncture and incision of intestines distended from peritonitis. In concluding this communication I propose to narrate a third case of a different type of diffuse peritonitis, which also recovered, and which incidentally illustrates the value of secondary intestinal drainage—that is to say, drainage established many hours after the laparotomy.

I have kept it apart from the others because it is of a different character, and because it was not operated upon in quite the same way. In it the peritonitis was

<sup>1</sup> *E.g.* see Mr. Croft's case, 'Clin. Soc. Trans.,' 1888, p. 254.

<sup>2</sup> 'Annales de Gynécologie,' 1889; also 'Brit. Med. Journ.,' October 5th, 1889, p. 778.

<sup>3</sup> My friend Dr. Tunnicliffe has pointed out to me that lime salts are essential for the integrity of living protoplasmic cells. Dr. Ringer has shown these salts to be present in New River water. An irrigating fluid would be best prepared by making normal saline solution '6 per cent., and then adding enough bibasic phosphate of lime to make it slightly milky.

accompanied by more adhesion around its starting-point, which was probably the vermiform appendix, and with much more pus in the pelvis. But it was without definite limits, and had involved the serous coat of the small intestines and mesentery, and of all the lower part of the abdomen. The coils of the intestines adhered very slightly to each other, and were quite easily separated with the finger. The details of the case were briefly as follows.

The patient was a postman aged twenty-five years. His illness began suddenly upon June 3rd, 1894, without premonitory symptoms. He was seized with violent pains in the abdomen, his bowels ceased to act, and he had occasional vomiting. He was treated with opium and a liquid diet. I saw him upon June 11th in the Great Northern Hospital. His temperature was  $100.2^{\circ}$  F., where it remained until the 13th, when it fell to  $98.6^{\circ}$  F. Throughout the rest of his illness it never rose beyond  $99.4^{\circ}$  F. His pulse was 88, and never exceeded 96 per minute. He was exceedingly ill, with an anxious expression, and was suffering great pain. The abdomen was tense, and did not move with respiration. In the right iliac region it was more distended than elsewhere, and there was an induration about the cæcum, with slight œdema and redness of the skin. It was obvious that he was suffering from typhlitis, and, inasmuch as I have seen similar cases recede, leeches were applied, and afterwards warm fomentations. During the 12th, 13th, and 14th the iliac swelling disappeared, but his condition became decidedly worse. The abdomen was more distended. The bowels did not act after the enemas, and it was questionable whether any flatus escaped. The pain was most severe in the lower abdomen, and was only relieved by opium. Moreover a tense fluctuating swelling was felt in the recto-vesical pouch. As it was obvious that the patient would die if unrelieved, on June 15th I opened the abdomen in the right linea semilunaris with the idea of finding the vermiform appendix, and, if necessary,

of removing it, and afterwards exploring and emptying the fluid in the pelvis. The tissues through which I cut were inflamed, and the parietal peritoneum could not be recognised. A substance was found which was supposed to be it, but an incision led into the cæcum. At this situation all the intestines were adherent, and they bled and were lacerated by the gentlest touch. The opening in the cæcum was therefore repaired with Lembert's sutures, and the abdominal wound was closed. A fresh incision was made in the linea alba. The peritoneum was thick and hard to recognise. In its interior the intestines were found acutely inflamed and covered with flakes of lymph. The peritonitis could be traced much higher than the wound, and had no definite limits. No firm adhesions were met with, and the various coils easily parted before the finger. About two pints of purulent and stinking fluid had collected in the pelvis. The vermiform appendix could neither be seen nor felt, and as there was much oozing of blood from the inflamed serous membrane, and as the man was in a precarious state, the abdomen was thoroughly washed with some gallons of hot water, and a glass drainage-tube was inserted into Douglas's pouch. The intestines were distended, but I did not puncture them, because, after the evacuation of the purulent fluid from the pelvis, they seemed to have enough room. He soon rallied under the usual remedies, and flatus was passed, both by a long tube and then naturally. The discharge sucked from the drainage-tube was purulent but not feculent. On June 18th the glass tube was changed for one of india rubber. The same night he again had a violent abdominal pain with a fresh outburst of the peritonitis. His abdomen became distended again, but fortunately his wound in the cæcum gave way. A quantity of flatus and fæces escaped, and his symptoms were gradually relieved. After this his bowels acted, although a good deal escaped by the fæcal fistula. By the end of July the median wound had nearly closed. On August 1st I closed the fæcal fistula by dissecting

the cæcum from the edges of the wound, and inverting the aperture with a double row of silk sutures. I was astonished on this occasion to find that the adhesions, which before had been troublesome, had almost disappeared. Indeed, after the cæcum had been freed from the edges of the wound it was easily drawn out, and many of the neighbouring coils of intestine came into view. They were unadherent, and looked quite healthy,—very different from their condition when seen six weeks before. As there was some purulent infiltration of the abdominal walls the wound was not closed. The sinuses were opened, the whole dusted with iodoform, and packed with iodoform gauze. The fistula never leaked again, and the wound speedily healed by granulation, so that the patient was discharged on September 3rd.

It is clear that this man owed more to good fortune than to surgery. I think he would have died if it had not been for the accidental opening into the cæcum. The formation of such an opening for the relief of the distension and obstruction which accompanies peritonitis has, as I have already said, been especially advocated by Senn and Greig Smith. The foregoing case seems to illustrate the soundness of their views. My colleague, Mr. Bruce Clarke, has recently had a case in which an opening into the small intestine saved a patient who had peritonitis after ovariectomy. I doubt not that many others could be adduced.

I have only to add a word upon the treatment of the intestines in this case. Had it been one of localised septic peritonitis care would have been taken to leave them undisturbed. In diffuse septic peritonitis I think it is advantageous to separate the coils from each other. In making post-mortem examinations after death from that disease, I have found much lymph and purulent fluid amongst them and amidst the folds of the mesentery. In my haste I could not tell whether this was so in the present instance. In another case, which ended fatally by a sudden syncope a few hours after the operation, I

found a great many collections of purulent fluid hidden away by the intestines and mesentery. Others have noted the same thing.<sup>1</sup> Further, although the adhesions are slight, yet they are extensive, and might prevent the recommencement of the peristaltic movements. Throughout the foregoing paper I have not referred to the pathology of diffuse septic peritonitis. Doubtless this term includes peritonitis which is due to many kinds of infection. It is evident, however, that some of these are amenable to surgical treatment.

<sup>1</sup> *E.g.* see a remarkable case by Messrs. Hawkins-Ambler and Knaggs, "A Case of Acute Diffuse Suppurative Peritonitis successfully treated by Laparotomy and Drainage," &c., 'Clin. Soc. Trans.,' vol. xxiii, 1890, p. 180.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. vii, p. 3.)



# A CASE OF ACTINOMYCOSIS EXTENSIVELY INVOLVING THE SKIN.

BY

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THE observation I have to submit is confessedly an imperfect one, and deals only with the *clinical* aspects of a case of extreme rarity, interesting from many other points of view. One of my reasons for bringing the matter forward even in this imperfect form is that the greater number of English contributions to our knowledge of the subject are embodied in the 'Transactions' of this Society; another is that I am in a position to show the patient to the Fellows while still alive, and presenting a condition hitherto unobserved in this country, and—as far as I have been able to ascertain—undepicted in any country. Circumstances, to which I shall subsequently allude, prevent the further investigation of the case from other than the clinical standpoint; but there can be little doubt that its fatal termination is inevitable, when the observation will be completed by a pathological and mycological report by my friend Dr. Wethered.

CASE.—Samuel H—, æt. 13, a schoolboy, presented himself at the Skin Department of the Middlesex

Hospital on September 28th, 1894. He was the subject of a skin eruption, with the appearance of which I was not familiar; and on account of this, as well as his obvious general ill-health, he was admitted to the hospital the same day, and charge of him was kindly transferred to me by my colleague Dr. Coupland.

*Family history.*—His father, mother, seven brothers, and three sisters are all alive and well. No trace of tuberculosis is to be discovered.

*Previous history.*—The patient has not suffered from the usual diseases of childhood, and indeed had never had a day's sickness until November, 1893, when he had an attack of mild pleurisy (?), for which he was confined to his room for two days only. He recovered completely, and remained well till the evening of Thursday, February 22nd, 1894, when, on retiring to bed, he noticed slight pain in the right side of the chest. This during the night gradually increased, and was accompanied by great difficulty of breathing. The following morning he felt better and went to school, but being again taken ill he was sent home, and was admitted to the hospital on Saturday, February 24th, suffering from shooting pain in the right side of the chest, increased by taking a deep breath, with a temperature of 101° F., pulse 96, and respirations 20 per minute.

The following is an epitomised account of his condition and progress while under the care of Dr. Kingston Fowler:

There were distinct diminution of movement, dulness, and absence of vocal fremitus on the right side of the chest up to the level of the fourth rib in front, above which the percussion note was skodaic. Below the level of the fourth rib the breath sounds and vocal resonance were greatly impaired, and bronchophony was present at the fourth rib. Similar physical signs were present over the corresponding portion of the back. The cardiac apex pulsated in the fourth and fifth interspaces in the nipple line. There was a dubious systolic apex murmur. Otherwise



the cardiac physical signs were normal. Appetite was poor, and there was some irregularity of the bowels. The urine presented no abnormality. It is specially to be noticed that *he is described as a well-nourished boy.*

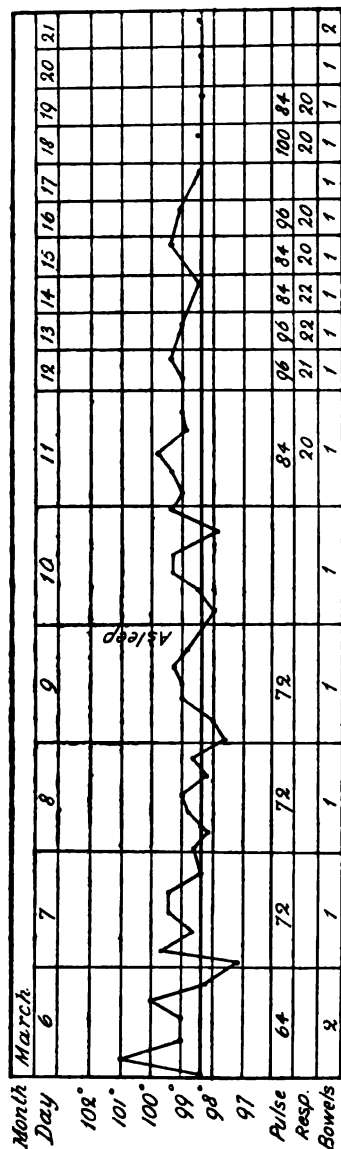
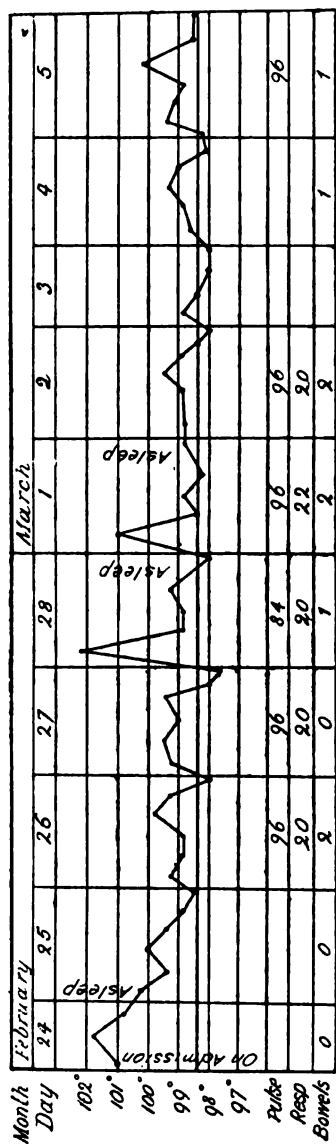
The subsequent progress of the case was such as to give rise to no suspicion that it was other than one of ordinary pleurisy with serous effusion, moderate in amount. The fluid gradually subsided, friction-rub was heard over the area previously occupied by fluid, and the patient was discharged from the hospital apparently convalescent on March 21st. For a few days previous to his discharge he complained of a pain in his right hip for which no cause could be discovered.

Appended (next page) is his temperature chart during this stay in hospital.

He was readmitted to a surgical ward in the hospital on April 3rd, on account of pain in the right hip and tenderness in the left. I am indebted to Mr. Hulke for permission to make use of his notes. Behind the right trochanter, in addition to a sensitive exostosis, there was a small swelling which fluctuated indistinctly, and there was a small, flat, tender, semi-fluctuating swelling over the seventh and eighth right costo-chondral articulations. Ten days later, after continued poulticing, these abscesses pointing were opened, but very little pus escaped. A week afterwards a similar abscess over the left ilium was also opened. On April 25th solid nitrate of silver was applied to "redundant granulations;" but the case did not progress favourably, so that on June 10th recourse was had to free scraping, when it was noted that the surrounding skin was extensively undermined. The boy was discharged to the Convalescent Home at Eastbourne on July 3rd, the ulcers not quite healed.

At Eastbourne rapid progress in the disease as affecting the skin occurred. Fresh lesions appeared on the front of the chest close to those previously referred to, and a similar eruption developed over the right loin. Several of the supposed abscesses were opened: but his

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general condition becoming more and more grave, a hacking cough having developed, and marked emaciation having set in, he was once again brought to the hospital and referred to me at the Out-patient Skin Department.

*State on admission.*—The patient is a dark-haired and sallow-complexioned boy, in a state of extreme weakness and emaciation, complaining of a hacking cough, and of peculiar ulcers on the back, chest, and hips. He has a hectic malar flush, and is said occasionally to sweat at night.

The most striking feature in the case, is an extensive and unusual neoplastic and ulcerative condition of the skin, the characters of which are well seen in the drawing<sup>1</sup> (Plate I).

*Description of the skin lesions.*—The skin of the back presents an extensive tract of disease, almost entirely confined to the right side. It extends vertically from the level of the seventh dorsal spine to the level of the posterior part of the crest of the ilium, a distance of nine inches. Transversely it extends from about one inch to the left of the spinal column (which is markedly curved, with its concavity towards the right side) a total distance of eight inches forwards, to terminate in the right lumbar region in a vertical line from the anterior superior iliac spine, and one inch and a half above it. The most striking feature of the eruption is constituted by enormous, livid, fleshy, sarcomatous-looking outgrowths of mottled purplish-red and yellow colour, numbering in all twenty-four, and varying in size from a split pea to a great prominent bossy mass, rising abruptly for more than three eighths of an inch above the level of the surrounding skin, and measuring four inches transversely by one inch and a half vertically at its broadest point. This mass lies transversely in the lumbar region, its lower margin being parallel to and two inches above the crest of the ilium.

<sup>1</sup> This was taken three days after admission, when the lesions had been cleared of crust and scab by appropriate means.

The larger tumours are made up by the fusion and coalescence of smaller ones, indicated by slight transverse bands in the case of the more cylindrical masses, and giving them a sort of moniliform appearance; while deeper fossæ indicate the lines of fusion of the more crescentic groups. To touch, all the growths are extremely soft, pulpy and fluctuating, and they are not tender. The skin over them is stretched, thinned, glistening, semi-transparent, and its blood-vessels dilated. Over the central part of the largest growth and underneath a portion of intact epidermis is a small recent hæmorrhage. Every growth presents at least one, and usually several small crateriform ulcerative openings, from which a clear, rather sticky fluid constantly exudes and trickles down the back in large quantities. In each of these ulcerated surfaces there is an accumulation of purulent fluid of pale sulphur-yellow colour, which is seen to contain innumerable tiny granular specks, which become much more obvious when examined with a magnifying glass. Over the larger growths the ulcerative lesions have run together to form considerable superficial discharging areas, at the margin of which there are small granular masses obviously made up of the specks described. Nowhere are there deep, hard-edged, or punched-out ragged ulcers comparable to those resulting from the breaking down of syphilitic or tubercular gummata.

The skin from which the tumours spring is of a deep purplish-brown colour, unaltered by pressure; and, in marked contrast to the growths themselves, is hard, brawny and tender to touch. This condition extends an inch and a half further forward than the anterior extremity of the largest neoplastic mass. The margin of the discoloured skin is pretty abruptly defined, but over the left side the skin exhibits some dappled brownish maculation, the colour of which is unaltered by pressure, and the signification of which is dubious, as the skin there is in every other respect normal. On the other

hand, the skin over the affected area already delimited, which is neither the seat of tumour-growth nor of pigmentation, although of normal colour, is found on palpation to be cedematous, boggy, and deeply undermined, as digital pressure at a considerable distance from the growths produces an outflow at the points of ulceration of the gummy fluid and purulent secretion containing the yellow granules referred to.

Over the front of the right side of the chest, from the fourth to the eighth rib, are three composite patches of disease, similar in general character to those already described; but having discharged their contents they are now passive, being for the most part converted into deep brown pigmented, sunken cicatrices. One of these patches was scraped in June.

Over the great trochanter of the right femur there are three similar pigmented scars, but they still show signs of activity and breaking down, and are very tender to touch; while over the centre of the left ilium is a raised ulcerating growth, similar to those described in detail on the back. (This is not indicated in the drawing.)

The presence of the yellow granular bodies referred to at once gave rise to the suspicion that the case was one of actinomycosis, and this diagnosis was readily confirmed by a microscopic examination of some of them without further preparation, when the characteristic club-shaped bodies were readily discerned.

Physical examination of the chest revealed the following conditions, but was carried out with difficulty owing to the extreme weakness of the boy.

There was marked retraction of the right side of the chest, the spinal column being notably curved, and the angle of the scapula tilted towards the vertebræ. Over the whole lower lobe of the right lung movement was *nil*, the percussion note was dull and breath sounds absent. Over the right upper lobe breath sounds were present, and rather bronchial in quality, but no adventitious sounds were present.

Over the whole of the left lung there was exaggerated breathing, but no other abnormality. Respiration was at the rate of 86 per minute. The cardiac apex was displaced outwards, and its beat most marked in the fifth interspace one inch outside the nipple line. The sounds were free from bruit; the pulse was 130 per minute. The liver could be felt to extend downwards with inspiration nearly to the level of the umbilicus, and by its obvious enlargement caused a prominence of the lower part of the anterior and lateral chest wall. Over the right side of the chest below the level of the third rib were several dilated veins, the blood in which coursed upwards towards the axilla.

The urine was acid, of specific gravity 1016, and contained a large amount of albumen and a copious deposit of lithates, but no casts. The tongue was clean. Appetite and digestion were good, and the bowels loose, 1—2 motions daily. His weight was 3 stone 6 lbs. He was ordered one thyroid tabloid of five grains of the gland daily, on the strength of very marked improvement under such treatment of a suspected case of actinomycosis of the cheek then and now under observation.

On the establishment of the diagnosis the following *history bearing upon the probable source of infection* was ascertained:

The patient's father was until Christmas, 1893, the foreman in charge of large stables in Baker Street in connection with an undertaker's establishment, and the family lived on the second floor above the stables, and had done so for nineteen years. The children played in the yard and had free access to the stables. Only two cases of fatal illness, however, had occurred in the stables during their stay: one horse died of "rupture of the bowel" two years ago; the other was slaughtered for "canker of the frog" four years ago. This latter horse *fourteen years ago* had a severe abscess of the jaw, the characters of which were so peculiar that the

veterinary surgeon in attendance made a drawing of it. It is stated that "the whole cheek fell out," but he made a rapid recovery, and almost no disfigurement resulted. The horse continued to work, and was something of a favourite until his death four years ago.

I am informed by Mr. J. A. W. Dollar, who attended the animal fourteen years ago, who made the drawing referred to, and to whom I am indebted for much valuable information regarding the disease, that the case was certainly not one of actinomycosis. A full consideration of the facts, even with our knowledge of the extremely prolonged incubation and latency of some cases, had already convinced me that there could be no direct connection between the affection of the horse and that of the child.

The *progress* of the case may be briefly epitomised.

The thyroid tabloids were increased to two daily on October 10th. For a few days he suffered from troublesome cough with copious mucoid expectoration, in which neither ray fungus nor tubercle bacilli were present.

The pulse-rate remained rapid, varying from 120 to 160 per minute. His respiration was also rapid—30 to 44 per minute—although he had no subjective dyspnoea. His temperature was generally subnormal, but occasionally rose above 99° F. in the evening. He had no night sweats. He gained in weight, and his general condition became decidedly more satisfactory than on admission. The condition of his skin underwent at first but little modification; the growths did not ulcerate beyond the extent figured in the water-colour drawing, although the discharge from them was abundant, and sometimes sufficiently so to soak through the dressings. It maintained the same gummy character as on admission, and was never markedly purulent or offensive. *Actinomyces* could always be easily discerned under the microscope. One fresh nodule about the size of a pea developed below the lesion on the right hip, which showed more

signs of activity than on admission. The bowels were loose, averaging two or three actions daily, the stools being liquid and pale. The urine remained highly albuminous. Repeated examination of the urine and fæces failed to reveal the presence of actinomyces. Boracic lotion was applied to the affected parts night and morning, which were afterwards dressed with boracic ointment and covered with "blue wool."

On October 16th the patient vomited repeatedly, and complained of faintness. The thyroid tabloids were therefore discontinued, with almost immediate cessation of the untoward symptoms.

The following day iodide of potassium was ordered in five-grain doses three times daily; and as distinct improvement ensued in a few days the dose was increased to ten grains on October 24th.

In the beginning of November he was allowed to sit up in an arm-chair, which he did without discomfort. Diarrhoea had ceased; he was taking his food excellently well, and had put on one pound in weight.

On November 7th the note ran as follows:—"The condition of the skin of the back has in some respects decidedly improved. The prominence of the tumours is decidedly less marked, and there are fewer ulcerative points. On the other hand a considerable number of small new growths have made their appearance, although not outside the area of skin considered as diseased; they develop upon normally coloured—not on pigmented—skin. The smallest are about the size of a pin's head, and first show themselves as yellow points; others are as large as peas, and are mottled, red and yellow. There is much less tenderness of the affected skin, which feels to the touch certainly less undermined. The boy's general condition is satisfactory, and he has gained  $2\frac{1}{2}$  lbs in weight—now weighing 3 st.  $8\frac{1}{2}$  lbs."

*Remarks.*—Although the most striking and exceptional features of the disease are now manifest upon the skin,



there can, I think, be little doubt that the case is an illustration of actinomycotic invasion through the respiratory tract. When the notorious difficulties of inoculating actinomycosis are borne in mind, it will not be a matter of surprise that few recorded cases are attributed to direct inoculation into the skin. Ponfick,<sup>1</sup> however, records his experience of the occurrence of inoculation, presumably from straw, in horses after castration. Leser<sup>2</sup> and Ressel<sup>3</sup> have collected a certain number of cases in the human subject, but their perusal convinces me that some are open to grave objection, many of the conditions described being referable rather to the introduction of tubercle bacilli or pyogenic organisms than to actinomyces. In eleven of these cases reported by Leser the disease was situated upon the leg; two were attributed to the patient's occupation—a ploughman and a farm labourer, one to a burn; in the rest the cause was undetermined.

Bertha,<sup>4</sup> however, records three indubitable instances (1) of inoculation of the hand while cutting oats with a sickle, (2) of the same part after threshing corn, and (3) of inoculation of the pharynx by an impacted oat grain; while Müller<sup>5</sup> records another of inoculation of the hand with a splinter of wood.

Whether or not the patient's attack of supposed "pleurisy" in November, 1893, may be considered as the first sign of his affection appears open to debate. On the one hand it may be noted that the child recovered perfectly, and remained well till February of this year; on the other, the extreme insidiousness and prolonged latency of the *first stage* of the disease when attacking the lung must be borne in mind. Israël,<sup>6</sup> who chiefly dwells upon the point, indeed says that the disease may run its

<sup>1</sup> Ponfick, 'Virchow's Archiv,' lxxxv, 1.

<sup>2</sup> Leser, 'Archiv f. klin. Chirurgie,' Halle, xxxix, 4, 1889.

<sup>3</sup> Ressel, 'Thesis,' Breslau, 1891.

<sup>4</sup> Bertha, 'Wiener med. Wochensch.,' 1888, No. 35.

<sup>5</sup> Müller, 'Bruns. Beiträge z. klin. Chirurg.,' Bd. iii, Heft 3, p. 355.

<sup>6</sup> Israël, 'Microparasites in Disease,' New Sydenham Soc., 1886, p. 476.

course without the patient feeling ill, without losing much of his power for work or suffering in appetite, digestion, or sleep ;" while, owing to the small size of the infiltrated patches, it may give rise to no physical signs.

There seems no room for doubt that the attack of acute pleurisy from which he suffered in February, 1894, was of actinomycotic origin, and personally I am inclined to think that it indicated the inauguration of the *second stage* of the disease, originating in and spreading from the lung, as in Dr. Markham Skerritt's<sup>1</sup> case. In their extremely admirable contribution to the literature of the subject, Dr. Douglas Powell, Mr. Godlee, Mr. Taylor, and Professor Crookshank<sup>2</sup> state their opinion that "actinomyces travel through lymphatics from the respiratory tract to the pleura, there vegetate in inflammatory tissues and secondarily invade compressed lungs," although this view seems to me hardly consonant with their other statement that the disease travels *across country*, in which opinion all foreign investigators agree, although, perhaps, they do not clothe their views in such sporting phraseology. The degree of shrinkage of the chest wall, out of proportion to the amount of fluid apparently present during the pleuritic attack, appears to me to support the view of the primary invasion of the lung, as indicating a condition similar to that recorded by Dr. Skerritt,<sup>3</sup> the affected lung in whose case is described as a dense mass of fibrous tissue. The non-purulent character of the expectoration, which does not contain actinomyces, the absence of moist sounds and the general passive character of the physical signs, seem to indicate that the disease in the lung and pleura is now inactive there.

There can, however, be no doubt that the disease has spread rapidly and extensively in other directions, and, as is its wont, without respect of tissue or organ. The fact that the boy was under close observation in the months of

<sup>1</sup> Skerritt, 'American Journ. of the Med. Sciences,' 1887.

<sup>2</sup> Powell, &c., 'Med.-Chir. Trans.,' 1889, vol. lxxii, p. 175.

<sup>3</sup> Loc. cit.

February and March is of value as enabling us to judge of this with great precision. At that time his liver was of normal size—now it reaches to the level of the umbilicus ; his cardiac apex is displaced one inch more to the left than formerly ; his urine was normal—now it is loaded with albumen ; he was a well-nourished boy—now he is a mere skeleton ; and his skin then presented no abnormality. I do not think that I assume too much in attributing both the enlargement of the liver and the presence of albumen in the urine to actinomycotic invasion ; nor is the latter assumption invalidated by our inability to detect actinomyces in the urine, for extension of the disease is known to be heralded by inflammatory changes of severe type and considerable extent.

The interest of the case centres in the remarkable manner in which the skin is involved, and to this point my principal remarks will be directed. It will be remembered that the first skin manifestations occurred, apparently simultaneously, in the beginning of April, over the right side of the chest and over the right great trochanter, being preceded in the latter situation by a deep-seated pain referred to the hip-joint, of about ten days' duration, possibly due to the exostosis noted there. The distance of this outlying islet of disease from the original seat of invasion suggests the possibility of a metastasis, contrary as the idea is to our knowledge of the usual mode of spread of actinomycosis.

Baumgarten<sup>1</sup> attaches the convenient epithet " cryptogenetic " to all cases of actinomycosis of the skin in which the path of infection cannot be with certainty determined, and suggests that the fungus may travel a considerable distance without producing morbid phenomena, or at most mere connective-tissue cell indurations.

On the other hand, the cropping up of the disease on the chest wall is in accordance with the statements of Israël,<sup>2</sup> who says, " It extends most frequently to the

<sup>1</sup> Baumgarten, ' Lehrbuch der path. Mykologie,' 12 Vorlesung.

<sup>2</sup> Loc. cit.

peripleural tissues of the chest wall, frequently involving the prevertebral tissues, more frequently posteriorly and laterally than anteriorly. If it starts from the lower part of the lung it may creep behind the costal insertion of the diaphragm downwards towards the posterior abdominal wall, spread there in the retro-peritoneal tissue, and from thence attack the ilio-psoas and quadratus lumborum muscles." The description I have given seems to confirm, as far as possible, Israël's description, —mere objective examination of the skin distinctly evidencing that the skin is invaded from beneath and much more extensively than inspection alone would lead one to suppose. The appearances presented by my case differ, however, in some respects from those cases recorded by Israël,<sup>1</sup> Majocchi,<sup>2</sup> Darier,<sup>3</sup> and others, which are thus epitomised by Dr. Radcliffe Crocker<sup>4</sup> in his short but admirable *résumé*; "the deep-seated actinomycotic tumour enlarges, suppurates, and as it approaches the surface the skin becomes red, livid, thinned, and undermined by suppuration, and fluctuating tumours are formed over the affected area, often with little or no pain."

Neither this description nor the single published coloured illustration (Neumann's 'Atlas,' Tafel xiii) of a case of actinomycosis of the jaw conveys any idea of the fleshy-looking masses presented by my patient. When the case first presented itself I was able at a glance to differentiate it from any recognised syphilitic or tubercular disease of the skin, and my thoughts turned rather towards some non-pigmented sarcoma or the fungating tumours of mycosis (*granuloma*) fungoides in its latest stage. The most striking features of difference were the pulpy softness and absence of pain or tenderness of the tumours, the absence of lymphatic enlargements, and more especially the manner of ulceration by small scattered discharging points

<sup>1</sup> Loc. cit.

<sup>2</sup> Majocchi, 'Bulletin d. Scien. Medic. di Bologna,' 1892, p. 517; 1 plate.

<sup>3</sup> Darier and Gautier, 'Annales de Derm.,' 1891, p. 449.

<sup>4</sup> Radcliffe Crocker, 'Diseases of the Skin,' 2nd edit., p. 840.

through epidermis for the most part intact, rather than by extensive necrosis *en masse*, resulting in the hideous, large, deep, fungating ulcers, with raised margins, characteristic of broken-down sarcomata or granulomata. The presence of typical yellow granules of course at once stamped the disease; but the abundant clear gummy discharge, although undescribed in other cases, appears to me also a distinguishing feature from similar conditions. It is a curious fact, illustrated by a case published by Legrain<sup>1</sup> and quoted by Crocker, that actinomyces are not readily found in all cases, and that inoculation experiments may be necessary to establish a diagnosis. I have under my care at the present time a woman presenting all the characteristic features of actinomycosis of the cheek, and which I diagnosed as such before this lad came under my observation, but in the scanty discharge from one softened nodule which broke I was unable to verify the existence of ray fungus. Unfortunately the scruples of this patient and her husband prevent me from having a portion of the disease excised, as also from applying surgical treatment, which would be peculiarly applicable to it, as the affection is strictly limited. This is the case referred to as having improved remarkably under thyroid treatment. In this connection reference may be made to the case reported by Billroth<sup>2</sup> as cured by fifteen injections of tuberculin, and to another cured by twenty-five injections of an extract prepared from the *Staphylococcus pyogenes aureus*. Ziegler<sup>3</sup> states that he has had similar results from tuberculin.

The treatment of actinomycosis in cattle by large doses of iodide of potassium was first instituted by Professor Nocard,<sup>4</sup> of Alfort, in 1885, and employed in the same year by Professor Thomassen,<sup>5</sup> of Utrecht, who

<sup>1</sup> Legrain, 'Annales de Derm.,' 1891, p. 772.

<sup>2</sup> Billroth, 'Wiener med. Presse,' 1891, p. 350.

<sup>3</sup> Ziegler, 'Münchener med. Wochenschr.,' 1892, 41.

<sup>4</sup> Nocard, 'Commun. to Soc. Central de Méd. vétérinaire,' 1885.

<sup>5</sup> Thomassen, 'Journ. of Comp. Path.,' 1892.

reported eighty consecutive successful cases. These results have been confirmed by Professor Ostertag,<sup>1</sup> of Berlin; and in Chicago, where 71 out of 185 affected oxen were cured by the drug.

In the human subject Netter<sup>2</sup> records the "prompt recovery" of one case of actinomycosis of the pleura and post-mediastinal tissues, and of two cases of actinomycotic pleurisy. A typical case affecting the jaw was cured in three months by Buzzi and Galli-Valerio.<sup>3</sup> As the ray fungus flourishes on gelatine containing 1 per cent. of iodide of potassium, it seems probable that the drug is not directly destructive of the organism, but acts by augmenting tissue resistance. In my case the result so far has been very encouraging, although I have not yet ventured upon the large doses recommended. At all events, surgical interference, which was suggested on his admission to hospital, was precluded by the lad's extreme weakness, and by the wide extent of disease, not only on the skin, but in internal organs.

#### *Demonstration of Actinomyces.*

The small yellow bodies removed from the thin pus exuding from the cutaneous lesions, and examined directly under the microscope, presented the characteristic club formation.

Efforts were made by Dr. Wethered to obtain cover-glass preparations in the following ways:

1. Stained in Ehrlich-Biondi solution and washed in water; unsuccessful.

2. Stained in picro-carmin and methylene blue; unsuccessful.

3. Stained in orange rubine, washed in water, dehydrated in absolute alcohol, and then the Weigert modification of Gram's method applied.

<sup>1</sup> Ostertag, 'Möllers Lehrbuch des spez. Chirurgie f. Thierärzte.'

<sup>2</sup> Netter, 'Tribune Med.,' 1893, No. 46.

<sup>3</sup> Buzzi and Galli-Valerio, abstracted in 'Therap. Gazette,' 1894, p. 35.

These specimens were partially successful; the mycelium was well seen, but the clubs were not. The chief reasons of failure, apparently, were, firstly, that the fungus was too thick, and when pressed out its form was entirely destroyed; secondly, that drying on the cover-glasses spoiled the delicate club formation, leaving the denser mycelium.

*Postscript.*—January 17th, 1895.—Although the lad's progress has on the whole been satisfactory, it has not been so rapid as was at one time hoped, owing probably to his inability to take doses of iodide of potassium larger than fifteen grains, which either cause sickness or severe bronchial catarrh. The remedy can be taken, however, in smaller doses, with occasional intermissions. The skin lesions have markedly improved, almost all having "withered" and flattened down to the general skin level. In the discharge from the few which are still ulcerating, ray fungus is still present but difficult to find. The amount of albumen in the urine is practically unchanged, but he now weighs 3 st. 13 lbs., and his general condition is correspondingly improved.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. vii, p. 12.)

#### DESCRIPTION OF PLATE I.

**A Case of Actinomycosis extensively involving the Skin.**  
(J. J. PRINGLE.)

Shows fleshy-like outgrowths with gummy fluid trickling from them and yellow discharge containing ray fungus. The intermediate and outlying skin is deeply pigmented.

The dappled pigmentation over the left side of the back is somewhat exaggerated.







# THE TREATMENT OF RESPIRATORY AFFECTIONS

BY MEANS OF

## LARGE MEDICINAL INJECTIONS THROUGH THE LARYNX.

BY

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(COMMUNICATED BY MR. WALTER WHITEHEAD.)

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To Professor A. Rosenberg, of Vienna, is due the credit of originating the method of treating pulmonary and laryngeal phthisis by the direct injection of medicinal fluids into the larynx and trachea through the glottis. His first paper on the subject was published in the 'Berliner klinische Wochenschrift' in 1885, and a more extended report on cases in the same journal in 1887.

Experiments to show its practicability, and the rapid absorption of which the pulmonary tissues are capable, have been made by Sehrwald and Reichert. The former ('Practitioner,' vol. xxxvii, 1886) injected the fluids "percutaneously," i. e. through an artificial opening in the trachea of dogs; and the result of his experiments showed—

1. That large quantities of fluid could be injected without discomfort.

2. That the fluids not only passed into the alveoli, permeating the surrounding tissues, but also reached the peribronchial and pleural fibrous structures, and even penetrated the cartilages themselves.

3. That the lungs absorb more rapidly than the digestive tract, or even the subcutaneous tissue, the rapidity of absorption corresponding with the extent of the absorbing surface: so much so, indeed, that the lung of a dog can absorb four times its own weight in less than five days.

4. That medicines thus introduced act in smaller doses, and more rapidly, than when introduced in any other way.

Reichert's experiments, made on calves and sheep, proved that the fluid injected found its way even into the finest bronchioles.

The 'Edinburgh Medical Journal' of January, 1888, contains an interesting report from Dr. A. Behag; and the method subsequently received considerable attention in Scotland from Drs. Jamieson, Downie, and others, who reported cases in the Edinburgh and Glasgow medical journals. Dr. Byrom Bramwell also reported two or three cases in his 'Clinical Studies' (June, 1889).

Rosenberg had recommended a solution of menthol, on which he relied because of its twofold (anti-bacillary and anæsthetic) action, in olive oil; and this solution, in varying strengths and doses, is the remedy used and reported on by the practitioners I have referred to. The cases treated appear to have been pulmonary phthisis and ulcerations of the larynx.

The last authority of importance to whom I shall refer, is Sir T. Grainger Stewart, who reported ('British Medical Journal,' vol. i, 1893) two cases of bronchiectasis treated with good results. He used a solution of menthol and guaiacol in olive oil.

Notwithstanding this publicity, and the favourable

opinion expressed by those who have tried it, I think I am justified in saying that to the great bulk of practitioners the treatment is unknown. This is my justification for asking the Royal Medical and Chirurgical Society to affix the stamp of its approval to a method which, in my humble opinion, opens up an entirely new field for pulmonary medication, and by a method eminently rational.

To bring the remedial agent into direct contact with the diseased part is a principle so well recognised in surgery, that it should require no argument to prove the *rationale* of extending its application to diseases of the respiratory system. Can anything be more faulty than our present system of attempting to render "aseptic" a pulmonary cavity—filled with tenacious muco-pus, and inhabited by myriads of bacilli—by a vapour, not a tenth part of which probably ever reaches the affected parts? I freely admit that improvement often follows the constant inhalation of the vapour of antiseptic drugs. But who would attempt the treatment of a spinal abscess by a "vapour" if he could wash it out first, and then send in his antiseptic solution or fill it with aseptic absorbent?

We are not yet in a position to attempt, with any hope of success, the washing out of a pulmonary cavity, but instead of the vapour or "smell" of an antiseptic we can send in the antiseptic itself.

If an ordinary abscess cavity were quite the same as a lung cavity, this would not be so much of an improvement in treatment. There is, however, some difference.

*A lung cavity does its best to empty itself*; not so an abscess. The persistent cough which accompanies it forces out its contents; and by suitable injections we can not alone aid this effort of nature, but make a reasonable attempt to render *less actively* septic that which remains behind.

*Is the treatment by intra-tracheal injection easily practicable?*

All the writers on the subject answer in the affirma-

tive. I think, however, that much practice is required to do it in a satisfactory manner. I have given nearly 4,000 injections, and thought myself an adept after the first dozen. I no longer think so. As in vaccination, or many other simple "operations," we always think ourselves perfect, yet practice reveals to us our imperfections. I shall, however, recur to this point.

I am not aware of any published cases showing the effects of intra-tracheal treatment in other diseases than phthisis, laryngeal ulcer, and bronchiectasis; but during the past eighteen months I have tried it in other pulmonary diseases, and with the most satisfactory results.

I propose to group the cases, rather than take them in chronological order. As there is only one case of bronchiectasis, I take it first.

No. 12.—It was a case of old-standing disease—one of the earliest cases treated by me. Having Sir T. Grainger Stewart's paper before me, I expected good results. One drachm, and later  $1\frac{1}{4}$  drachms, of a solution—4 per cent. guaiacol, 12 per cent. menthol, in olive oil—were injected twice daily for a month, but no beneficial result followed. Observation of this case first suggested to me that olive oil was not the best vehicle in which to convey the medicinal agent to the lung. A demonstration of this was easy. I collected some of the dense expectoration from the case in a test-tube, closed with a cork at one end, and poured some of the oily solution on to it; after twenty-four hours the oil still remained at the top, whilst the lower stratum (which could be smelt by removing the cork) did not appear to contain either menthol or guaiacol. A glycerine solution at once suggested itself. My first experiences with glycerine solutions were, however, quite unsatisfactory. I could only note that *in a test-tube* it mixed better with expectorated material, and therefore presumably ought to bring the drugs (if it would only carry them) into closer contact with the diseased parts.

It is all very well to demonstrate, as Sehrwald and

Reichert have done, that the injected material can be found all through the lung tissues after an injection. So could it be found in the bladder. A few minutes after an intra-tracheal injection of turpentine I have noticed the characteristic smell of violets in the urine.

The simple tube experiment mentioned suggests that the finding of menthol in the bronchioles, &c., after the injection of an oily solution (not miscible with the bronchial secretions) is more probably due to a secondary effort at *excretion* (subsequent to absorption) so easily noticed in the kidneys; and this, although probably very beneficial, is, I take it, quite different from bringing the original strong solution into *direct contact* with the diseased parts.

That glycerine is the best solvent for remedies directed to pulmonary phthisis, bronchitis, or asthma, I have no doubt; but for months I did not succeed in finding a satisfactory solution. It is unnecessary to go into detail. I found that the ordinary "glycerines" are most irritating to the bronchial membrane. Price's distilled glycerine is not so. Menthol, guaiacol, creasote, benzosol, &c., can be kept in solution in glycerine by heat.

The consideration of case No. 12 and its lessons have drawn me from the path. I now propose to take up the cases of phthisis, and roughly place them in two groups: (A) with cavities; (B) with consolidation. The table of cases appended also shows those cases in the sputa of which bacilli were found; and I place the specimens before the Society.

(A) No. 1.—When this patient commenced treatment, in June, 1893, her right lung was one vast cavity; her sputum was loaded with bacilli. I can only say that she is still alive, and comes for injections two or three times a day; when she is unable to come she complains of being worse in every way—she loses appetite, the cough becomes more troublesome, and the pain is more acute. She has had at least 1450 injections; No. 1 solution (see page 50) appears to do her most good.

No. 2 may be regarded as a really satisfactory case. Her father and mother had both died of phthisis under my care. When she commenced treatment she had a small cavity in the right apex, and the sputum was loaded with bacilli. She has now regained flesh and lost her cough, and the cavity is apparently cicatrising. No. 1 solution appears to have done most good.

Cases 3, 4, 5, and 7 may be considered together. They were all cases of advanced phthisis with large cavities existing in one or both lungs. They were not improved, although they expressed themselves *relieved*, by the treatment.

(B) The cases of phthisis in which no cavity could be found show much more satisfactory results.

No. 8 is probably the most remarkable case. Her father and mother and one sister (out of two) have all died of phthisis. She was distinctly tuberculous, sweating and diarrhoea regularly alternating in attendance on a persistent hard cough. The diarrhoea was not checked, although everything was tried, till I commenced the injection of benzosol. She has gained much flesh, but a continuation of "fresh colds" (almost impossible to be avoided by a girl who leaves a hot woollen mill and walks four miles daily for her injection) always appears to block the path to recovery.

No. 9.—His mother died of senile phthisis, his father of cancer. When the patient first came for treatment he had recently had an attack of hæmoptysis; he had a persistent cough, and was losing flesh. Twenty injections appeared to cure his cough, and he has followed his occupation as a stonemason for the past twelve months. In this case the solution recommended by Sir T. Grainger Stewart—menthol, guaiacol, and oil—was used throughout.

No. 10 was a case of an almost exactly similar character. She consulted me a few days after an attack of hæmoptysis (which was preceded by cough and loss of weight). One month's daily injections cured her cough, whilst she gained 8 lbs. in weight. She has continued



well ever since. Both her parents died of phthisis. Treatment: Sir T. Grainger Stewart's solution; later No. 1 solution daily, at one sitting.

No. 11 was a case of consolidation of the right apex with asthma (which had existed four years). About twenty daily injections completely relieved the asthma. He thought himself cured, and I lost sight of him then. No. 1 solution was used in 3-drachm doses.

No. 10A unfortunately only shows the results of clumsy manipulation. Three of the patient's brothers had died of phthisis, one under my care. It was a typical case of early laryngeal tuberculosis with ulceration. The sputum pellets showed masses of bacilli, and he was rapidly losing flesh. I could not, however, convince the patient that there was anything seriously wrong with him, and portions of a few injections of Sir T. Grainger Stewart's solution having unfortunately found their way into the stomach and caused discomfort, the patient discontinued treatment. I have heard nothing of him since. (I shall refer to the results of injections finding their way to the stomach again.)

No. 21.—An advanced case of pulmonary phthisis with cavities, laryngeal ulceration, and severe dysphagia. Patient attends irregularly, but injections of 3 drachms of No. 1 solution on alternate days have almost completely relieved the throat symptoms. At first an injection was followed by loss of pain for eight hours; now the relief continues for forty-eight hours.

(Note.—This cumulative effect [as an anæsthetic] of menthol is referred to by Dr. Rosenberg in his second paper.)

In every case of phthisis treated (excepting 10A) the patients expressed themselves as much relieved by the treatment. Even in the fatal cases, relief of cough, pain, soreness in throat and sweating was always expressed. Excepting the case 10A, no patient ever sought other treatment when this failed, so satisfied were they with its reasonableness and effects.

In no case, however, can I claim a *cure* of phthisis in the third stage. Case 2 looks hopeful, but I am well aware how rapidly such cases relapse. I think, however, that cases 8, 9, 10, and 11 testify to important results in the early stages, so far bearing out the observations of Rosenberg, Behag, Downie, and others.

Three cases of phthisis remain to be mentioned; I shall refer to them under the head of hæmoptysis.

The other cases which follow show more satisfactory results for intra-tracheal treatment; for example:

No. 6. *Abcess of the lung*.—He came to my surgery, October, 1893, supported by his wife and a friend. The history was that he had had a cough for a year; three months since he had an attack of pneumonia. When I saw him his respirations were 40 per minute, temperature  $102^{\circ}$ , pulse 120. He was livid in the face, his left lung appeared to be rapidly softening in the lower two thirds, and he was spitting 38 oz. of red, stinking matter—mostly pus—in twenty-four hours.

I first injected 1 drachm of oil of turpentine (with an equal quantity of olive oil) daily for four days; then changed to terebene in equal strength. Many other combinations were tried during seven months of treatment, after which he returned to his work apparently "cured."

No. 13. *Chronic pneumonia*.—A boy æt. 14 had had two attacks of pneumonia. Three months after an imperfect "recovery" from the last attack he continued to expectorate large quantities of muco-pus, slightly tinged with blood. His respirations were 32, pulse 110, face livid, and shoulders much stooped. He was treated for four months, during which time he had 250 injections—turpentine, terebene, Sir T. Grainger Stewart's, and finally No. 1 solution being used. At the present time—four months after treatment has been discontinued—he appears perfectly well.

No. 14. *Asthma*.—This is a remarkable case. The patient had suffered from asthma almost continually for

twenty years. At the time the intra-tracheal treatment was commenced he had been using "Himrod's cure," four or five times a night, for some months. After the first half-dozen squirts he was enabled to discontinue its use; whilst twenty-four daily injections (last given April, 1894) appear to have "cured" him, as he has not had a single attack since.

The *expectorant* power of glycerine injected into the lung was well evidenced in this case. At first I injected, alternately, oil, solution No. 1, and a glycerine solution of menthol and benzosol. Both gave relief—the oil for four or five hours; but the injection of the glycerine solution was always followed by a copious *emptying* of the lungs, causing relief for twenty-four hours. This I think is another advantage of glycerine over oil.

No. 15.—A lady æt. 65 had suffered from asthma with bronchitis for over twenty years. She still suffers from bronchorrhœa; but she has not had a "bad night" since last May, when she finished her course of twenty-four daily injections, the same as used in the last case.

No. 16.—Third case of asthma; he was unable to work when I first tried the treatment. Ten daily injections (alternated as last) relieved him so much that he was able to take work at an iron foundry. Lately he has been coming every Sunday for an injection, which he says "opens his lungs" for the week.

No. 17.—There is nothing remarkable about this case of chronic bronchitis of an ordinary character. The patient had six injections only of No. 1 last May, and says that he has never been "bad" since.

Nos. 18, 19, and 20, laryngitis and aphonia, may be taken together. They were all treated with No. 4 solution (injected once daily through a nozzle with *side* openings), and were immediately "cured."

*Hæmoptysis*.—There remain Cases 22 and 23. No. 22 is a case of advanced phthisis, and 23 is in an early stage. In each hæmoptysis was the symptom treated. I place them, therefore, under that heading. In No. 23 severe

hæmorrhage occurred last March, and continued two or three days. It did not recur after an injection of 4 drachms of No. 2 solution. This is only mentioned as an example of the use of turpentine intra-tracheally. No. 23 is a better example. I was called to see him on October 19 last. At 5 a.m. that day he had suddenly expectorated  $\frac{3}{4}$  of a pint of bright red blood. When I saw him at 11 a.m. he had again commenced to spit copiously. Ergot, gallic acid, &c., were prescribed with temporary relief, but the following day he was again spitting blood freely. I then injected 2 drachms of oil of turpentine with an equal quantity of olive oil; in a few minutes the bleeding ceased, and has not recurred. The injection was repeated on the two days following.

P.S.—A third case of hæmoptysis has since been treated by injection of turpentine. S. S—, æt. 48, had a severe hæmorrhage on November 16th. In spite of a strong acid astringent which had relieved him on two previous occasions, he was still spitting blood profusely when I saw him on the night of the 18th. Two drachms of turpentine in olive oil were injected, after which no more *red* blood was expectorated. The injection was repeated on the two following days. As in the other cases mentioned, no inconvenience was caused by the treatment.

It is unnecessary for me to describe the *modus operandi*, but I would venture to differ from what has been written on the question of dosage. Neither Rosenberg nor Downie states exactly what quantity he injects at each "squirt," though they mention  $\frac{1}{2}$  to 1 drachm at a *sitting*. Bramwell suggests 15 minims at once. I have found it best generally to empty a syringe holding about 100 minims at *each squirt*, and to repeat this 100-minims two or three times at each sitting.

Differences will be found, strange to say, in the position of the rima glottidis with regard to the base of the

tongue, and occasionally one finds a patient in whom the epiglottis habitually forms an acute angle with the rima.

The size of the rima, again, differs much in different patients. To learn my way about, therefore, I always use a laryngeal reflector for the first few injections, and afterwards I work without one.

If, when the injection is delivered, the patient be directed to incline or lie on either side, it appears probable that the bulk of the fluid will gravitate in the desired direction. I have tried to inject patients whilst they were leaning to one side, but the result was unsatisfactory.

At first I employ tongue traction, but after a few injections the patients do it, holding the tongue with their own pocket handkerchiefs, thus diminishing the risk of infection.

After each sitting the syringe should be washed out with a strong solution of carbolic acid.

*If the operation be properly performed* no inconvenience results—neither asphyxia (which has many times been suggested to me as *impossible* to avoid), pain, nor even cough. At first, I confess, my patients grumbled much, but after a painstaking experience of a few dozen injections, complaints became less frequent.

Three mistakes are liable to occur :

1. Touching the fauces, base of tongue, epiglottis or rima glottidis with the nozzle of the syringe.
2. Squirting down the œsophagus.
3. Squirting during expiration.

If the first happen, efforts at vomiting usually result. If the second, gastric pain occurs, with unpleasant eructations when menthol or guaiacol is used. If the third, a feeling of suffocation follows, whilst some or all of the fluid intended for the lungs is returned into the mouth. These unpleasant results can best be avoided by great care in passing the tube through the mouth and fauces. When the nozzle of the tube is seen to be behind and below the

top of the epiglottis, the tube should be rapidly passed into the larynx, and should fit the curve of the base of the tongue, and lie tightly against it, thus *fixing the epiglottis* and *preventing spasm*. The squirt should be delivered like lightning—either with inspiration, or during a slightly prolonged interval following expiration (except when used for laryngeal affections).

If the operation be properly performed, the taste of the fluid injected should not be perceived by the patient.

The object of this paper is rather to advocate a *principle of treatment* than any special drugs or combinations. I have tried many combinations during the past eighteen months, which it is useless to describe in detail. I think almost anything can be injected: water, olive oil, or glycerine can be used in quantities of 4 or even 6 drachms twice or three times a day without discomfort.

Instead of describing the many combinations tried in each case, it will perhaps be more satisfactory to mention those from which I think the best results were obtained, with the conditions to which they are applicable.

#### SOLUTIONS FOR INTRA-TRACHEAL INJECTION.

No. 1. *Phthisis, ulceration of larynx, asthma*.—(A) Benzozol 24 grains, rectified spirit  $\frac{1}{2}$  drachm; mix and add Price's distilled glycerine  $\frac{1}{2}$  ounce; dissolve by heat. (B) Menthol 24 grains, rectified spirit  $\frac{1}{2}$  drachm, glycerine (as above)  $\frac{1}{2}$  ounce; dissolve by heat. Mix A and B, and keep at a temperature of 90° Fahr. or warm before using.

*Dose*: 2 drachms twice or three times a day.

No. 2. *Hæmoptysis*.—Oil of turpentine, olive oil, of each 1 ounce; mix. Inject 4 drachms daily for three days.

No. 3. *Abscess of lung, bronchorrhœa, &c.*—Terebene (Cleaver's ordinary), olive oil, of each 1 ounce; mix. Inject 3 or 4 drachms daily, or use alternately with No. 1.

*No. 4. Laryngitis, aphonia.*—Menthol 24 grains, olive oil  $\frac{1}{2}$  ounce; mix. Inject 3 or 4 drachms *slowly* once daily.

*No. 5. Asthma.*—Sir T. Grainger Stewart's solution: Menthol 10 parts, guaiacol 2 parts, olive oil 88 parts. Inject 2 or 3 drachms on alternate days with No. 1.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. vii, p. 20.)

*Appendix to Mr. Colin Campbell's paper.*

No.	Age and sex.	Disease.	Duration of treatment.	No. of injections given.	Bacilli present.	Result.
1	24, F.	Phthisis (cavity)	16 months	1450	Yes	Symptoms relieved; cavity undiminished.
2	25, F.	" "	14 "	370	"	Much improved in every way.
3	45, M.	" "	8 "	400	"	Dead.
4	43, F.	" "	1 month	30	"	Dead.
5	28, M.	" "	1 "	40	Yes	Dead.
6	51, M.	Abscess of lung	6 months	370	No	Recovered.
7	27, M.	Phthisis (cavity)	2 "	20	Yes	Dead.
8	26, F.	" "	14 "	370	"	Improved.
9	3, M.	" P	1 month	25	"	Recovered.
10	24, F.	" "	1 "	25	Yes	Recovered (P).
10A	27, M.	" "	4 days	4	"	Discontinued treatment.
11	27, M.	" Asthma	—	80	"	Improved.
12	64, M.	Bronchiectasis	1 month	20	"	No improvement.
13	64, M.	Chr. bronchitis	5 months	250	"	Recovered.
14	15, M.	Asthma	24 days	24	"	Recovered.
15	57, M.	"	24 "	24	"	Recovered.
16	F.	"	1 month	30	"	Recovered.
17	62, M.	Chr. bronchitis	1 week	6	"	Recovered.
18	37, M.	Laryngitis	6 days	6	"	Recovered.
19	80, F.	Laryngitis and aphonia	6 "	6	"	Recovered.
20	38, M.	Phthisis (cavity), ulceration of larynx	6 "	6	"	Recovered.
21	38, M.	Phthisis, hæmoptysis	14 "	24	Yes	Symptoms much relieved.
22	M.	Hæmoptysis	8 "	3	"	Hæmoptysis ceased immediately.
23	23, M.	Hæmoptysis	8 "	3	"	Hæmoptysis ceased immediately.
24	56, M.	Chr. bronchitis	12 "	12	"	Much improved.
25	46, M.	Hæmoptysis	4 "	4	"	Hæmoptysis ceased immediately.



# A YEAR'S EXPERIENCE OF THE USE OF SULPHUR IN SURGERY.

BY

W. ARBUTHNOT LANE, M.S.

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As more than a year has elapsed since I commenced to use sulphur in surgery, I venture to lay before this Society some record of the experience of its action which I have gained during that time, together with such conclusions as I have arrived at as to the best methods of its application.

It was my misfortune to lose a patient by iodoform poisoning. He had suffered from spinal caries in the lower part of the dorsal region, and a large superficial abscess had formed beneath the skin in the vicinity. The abscess was laid freely open and its walls scraped. A cavity in the chest near the carious vertebral bodies, which communicated with the superficial abscess by an opening between the ribs, was also cleared of its contents as thoroughly as possible. Then glycerine and iodoform emulsion was introduced into the cavity, and most of the incisions were closed by means of sutures, a drainage-tube being left in. I had adopted similar treatment on a great many occasions with considerable success, and in no instance had it been followed, as far as I knew, by any ill consequence attributable to the drug. On the day after the operation the patient's pulse became rapid, and during the night his mental condition attracted

some attention. Dr. Thomas Stevenson saw him, in consultation with me, and confirmed my suspicions that the patient was suffering from iodoform poisoning. Like all patients into whom iodoform has been introduced, his urine contained iodine in readily recognisable quantities. The wounds were at once opened up and freely irrigated, every remaining particle of iodoform being removed. In spite of this, and in spite of the introduction of a large quantity of normal saline solution into his circulation with the view of enabling him to excrete the poisonous material through his kidneys, the rate of his pulse, the mental depression and the delirium increased, and he died within four days of the operation.

It seems likely that this patient possessed a peculiar idiosyncrasy in regard to iodoform, as there was no variation from the modes of operation previously adopted to account in any way for the drug producing such dangerous symptoms. Though I felt I was free from blame for the unfortunate termination of the case, yet it caused me much distress and a feeling of distrust and uneasiness in the use of the drug. I could not help fearing that it soon might be my misfortune to experience another such disaster; and I spent much time in thinking of some material that would take the place of iodoform without possessing its poisonous properties. It would appear that iodoform produces but little if any effect upon organisms unless it is placed in contact with a raw living surface, when the living tissues break it up into its components, one at least of which is powerfully germicidal. It occurred to me that sulphur, which is able to check the growth of the *acarus* of scabies, *Tricophyton tonsurans*, *Microsporon furfur*, and other animalcula and micro-organisms, might, under the influence of raw living tissue, produce sulphurous acid in a quantity sufficient to diminish or even stop the growth of any organisms with which it might come in contact.

On consulting my friends as to the possibility of its being of service in this manner I received but little sup-

port. I was, however, comforted by the general assurance that at least the drug could do no harm, as, if sterilised, it would probably remain inert when introduced into an aseptic wound.

The first case for which I used it was that of a girl aged ten years, who was admitted under my care into the Hospital for Sick Children, suffering from very advanced and rapidly progressive hip-joint disease. I operated on September 19th, 1893, and found very extensive disease involving the head of the femur, the acetabulum, and the adjacent portions of the innominate bone, and associated with this, abscesses extending into the true pelvis, into the buttock, and down the thigh. There was no pocket or cavity in the neck of the femur in which I could pack a quantity of iodoform; and though pieces of necrosed bone were removed from the pelvis, no opportunity for such treatment offered itself there.<sup>1</sup> After clearing away the necrosed and caseous structures as well as I could, and after removing the contents and scraping the walls of the abscess cavities as thoroughly as their position would allow me, which in the case of the large collection in the true pelvis was of necessity done very imperfectly, I felt convinced that, if the case were left in this condition, the disease would return in a very short time as acutely as before, and that the patient would have derived very little benefit from the operation. I therefore introduced a quantity of sterilised precipitated sulphur into the wound, in the hope of its being of service in staying the development of the tubercle bacillus, and closed the incision, leaving in a drainage-tube. The limb was then put up in its position of rest, the inner margin of the foot forming with the vertical an angle of about 50°. At

<sup>1</sup> I may say in passing that this is the only use I now make of iodoform, namely, to plug cavities in bone with the solid drug. This very insoluble material is slowly removed, its place being taken by new bone. This method I described in the 'Lancet,' July 15th, 1893, in a paper entitled "One of the Best Applications of Iodoform in Surgery."

the end of forty-eight hours the tube was removed, when the odour of sulphuretted hydrogen was perceived.

The case followed the ordinary course of an aseptic wound till the 25th of October, when the temperature rose to  $100.5^{\circ}$ . The wound was dressed on the 26th, when the smell of sulphuretted hydrogen was more obvious. By the 30th, the edges of the incision, which had previously united, had broken down, became black and sloughy, and gas escaped freely. The patient was put under an anæsthetic, and the parts beneath the skin were freely exposed. It was then found that not only the skin, but also the fat, fascia, muscles, and bone were most extensively destroyed. Indeed, it was with the greatest satisfaction that I found the femoral artery pulsating in the depths of what seemed like one extensive slough. The soft broken-down tissues and the necrosed surfaces of bone were removed very carefully, and the wound was freed of any perceptible relic of unchanged sulphur.

It was again covered by an inhibitory dressing, and was irrigated daily with a dilute perchloride of mercury lotion. The remaining sloughs separated rapidly, and union took place in a most satisfactory manner, leaving a very useful firm limb.<sup>1</sup>

I gathered from this experience that sulphur, under the influence of recently incised living tissues, produced sulphurous acid, which rapidly changed to sulphuric acid by oxidation, and that this acid was formed in quantities sufficient to destroy not only any organisms with which it came into contact, but every living structure as well, whether muscle, bone, fat, fascia, or skin. It also was apparent that I had used this powerful drug in an excessive quantity, that I had left it in the wound too long, and that I was dealing with an agent which, if used properly, must be of the greatest service to the surgeon in dealing with many conditions which had

<sup>1</sup> This patient was readmitted under my care in 1894 with extensive tubercular disease of the ankle, which I erased successfully.

hitherto successfully resisted all his efforts. I soon learned to use the drug properly, and have arrived at the following conclusions :

1. Neither sulphur nor the products generated by its decomposition act prejudicially upon the life or health of the individual into whose body it is introduced.

2. If placed in contact with recently incised healthy structures, twenty-four hours is a sufficiently long period for it to render the part sterile. After using it in this manner it is necessary to leave the wound open, so that the sloughs which have formed may escape. In my earlier cases I used to irrigate the wounds at intervals with a dilute perchloride of mercury lotion, but now I prefer to pack with sterilised gauze.

3. If the recently incised or scraped surface be but poorly supplied with blood—as, for example, is the brawny edge of a carbuncle or that of a spreading gangrene of a limb—the sulphur may with advantage be left in contact with the tissues for a considerably longer period.

4. If the surface be a granulating one—such, for instance, as the wall of a chronic tubercular abscess—sulphur may be introduced daily, preferably as an emulsion with glycerine, without causing any considerable destruction of parts.

5. In such joints as are reached with difficulty—as, for instance, those of the tarsus, the sacro-iliac synchondrosis, &c.—the tubercular growth may be reached by diffusing sulphur and glycerine between the articular surfaces as often as may be necessary.

6. The presence of other organisms in a tubercular cavity, whether due to the surgeon or admitted by a spontaneous rupture of its walls, influences in no way the action of the drug, since it destroys all organisms, whether free in the cavity or intruding into the surrounding living structures forming its walls.

7. The action of the sulphur is painless.

8. The formation of new compounds caused by the action of living tissues upon such drugs as iodoform or sulphur, which, without such influence, are practically harmless to organisms, illustrates forcibly the fallacies that may arise from the attempts of bacteriologists to demonstrate the capacity of drugs as inhibitory or germicidal agents by means of experiments on dead cultivation media alone.

I cannot do better than relate briefly the histories of some of the cases in which I have used it.

A. B—, æt. 7 years, was admitted under my care into the Hospital for Sick Children, suffering from very advanced disease of the elbow-joint. I operated on September 22nd, 1893, and found the several bones most extensively diseased. This condition, with the very largely distributed boggy infiltration of the soft parts about the joint, rendered it extremely unlikely that a good result would follow an excision. Consequently, after removing the diseased bone and tissues as freely as I could, I introduced a quantity of sulphur. Much swelling ensued, and the part was again exposed on the seventh day after the operation. The changes found were similar to those described in the first case, though not so extensive, while the treatment adopted was the same. From this date the patient made an uninterrupted recovery, and has now a strong useful joint with moderate movement. I believe that a primary excision of the articulation would very probably have been followed by an amputation, but on this point it is obvious that I cannot speak with any certainty. As in the case first mentioned, the sulphur was left in for an unnecessarily long period.

A man æt. 30 was admitted into Guy's Hospital under my care on July 11th, 1894. He had been cleaning a window, when he fell forty feet, transfixing his forearm upon an iron spike of the area railings, from which he was suspended. The skin and muscles of the forearm were extensively lacerated along the whole of its length,

and portions of his coat-sleeve and shirt, which were both very dirty, were driven in among the pulped and lacerated muscles. Such portions of the muscles as were much mashed and fouled were removed. The ulnar artery was uninjured. The damage to the soft parts was so extensive and the fouling so considerable that I was convinced that, however thoroughly the parts might be washed with germicidal lotions, amputation would become necessary at no distant date.

Therefore, after cutting away some parts, cleaning up others, and removing all foreign materials present, gauze saturated with an emulsion of sulphur and glycerine was introduced everywhere in and between the lacerated tissues. This was removed at the end of twenty-four hours, when the wound was found to smell strongly of sulphuretted hydrogen, and the tissues to be covered by a soft black slough. Irrigation with dilute perchloride of mercury lotion was used daily, and the intervals in and between the lacerated muscles were packed with cyanide gauze. The slough soon separated, leaving a healthy granulating surface. The highest temperature recorded was  $100.6^{\circ}$  on the evening following the operation. I would repeat that, as far as my experience goes, this arm would have become foul very soon after being treated with the germicidal solutions we use for such purposes, and amputation would have been rendered imperative in consequence. He recovered with a very useful arm.

L. J—, æt. 12 years, was admitted into Guy's Hospital under my care on November 8th, 1893, suffering from tubercular disease of the base of the third metacarpal bone, and from extensive lupoid ulceration which covered the back of the hand and wrist. This had obviously resulted from an infection of the skin where the tubercular abscess about the base of the metacarpal bone opened externally.

The cavity in the bone was scraped clean and plugged:

firmly with iodoform. The extensive patch of lupus was scraped and covered with lint soaked in glycerine and sulphur emulsion. When this was removed at the end of twenty-four hours the surface was seen to be covered with a soft black slough which smelt strongly of sulphuretted hydrogen. This separated under the influence of hot boracic compresses, and on the clean surface a large single graft was placed. The patient was discharged with the sinus in the bone and the skin surface firmly healed. She experienced no pain from the action of the sulphur.

E. T—, æt. 17, was admitted into Guy's Hospital under my care on October 30th, 1893, suffering from tubercular disease of the tarsus, which was much swollen in the vicinity of the medio-tarsal joint, and of the articulations between the cuboid and cuneiform bones. Externally there was an opening as large as a sixpence, through which a probe could be passed deeply into soft caseous bone and granulation tissue.

The cavity in the cuboid was scraped out thoroughly, and much caseous material was removed from it and from adjacent bones. Glycerine and sulphur emulsion was introduced into the cavity and made to percolate into the several affected articulations, and on subsequent occasions small quantities were dropped into the sinus. The swelling of the foot rapidly subsided, and the patient was sent to a convalescent home with a depressed cicatrix occupying the position of the sinus. She had no pain or discomfort in the foot.

The amount of swelling and the very extensive evidence of disease of the joints and bones of the tarsus would have led me to perform a Syme's or Pirogoff's amputation if I had not been aware of the action of sulphur.

W. C—, æt. 11 years, was admitted into Guy's Hospital under my care on November 24th, 1893, suffering from extensive tubercular disease of the lumbar and sacral spine, discharging externally by several sinuses. An



attempt was made to get at the disease by extensive incisions, but without much success; gauze saturated with sulphur and glycerine was introduced, and the cavities were plugged as firmly as possible. This was removed on the following day, and sterilised iodoform gauze was substituted. A strong odour of sulphuretted hydrogen was given off from the wound, and this continued for a long time, owing to the fact that much of the sulphur must have been left in the irregular branching abscess cavities.

Before this operation the boy had been losing ground steadily, but almost immediately after the operation the improvement both locally and in the general condition was most remarkable. The cavities and sinuses soon closed, and the boy recovered perfectly. He was seen a fortnight ago, when he was leading a very active life and enjoying very good health, his cicatrices being white and firm.

As far as my experience goes such a result could not have been obtained in the time, if at all, by means of any other method with which I am familiar. But for the sulphur treatment I believe the boy would have died within six months.

F. G—, æt. 47 years, employed in the brewing trade, was admitted into Guy's Hospital under my care on August 20th, 1893, suffering from a lacerated wound of the front of the dorsum of the foot. It was fouled by dirt. He acknowledged that he had been a very free drinker for many years. The patient was put under an anæsthetic and the wound was thoroughly cleared of foreign material, all ragged tissue was cut away, and the wound as well as the rest of the foot was rendered as clean as possible by means of liquor potassæ, soap, and strong carbolic lotion. Cyanide gauze moistened with one in twenty carbolic lotion was applied.

When the dressing was removed at the end of forty-eight hours the edge of the wound and the tissues in its immediate vicinity were found to be gangrenous, while

the foot and leg were œdematous. The wound was again cleaned with strong carbolic lotion and repacked with cyanide gauze, as on the previous occasion, and over this dressing a large hot carbolic compress was applied and the limb was raised. Twenty-four hours later the gangrene had spread considerably, while the skin and subcutaneous tissues of the foot were very thick, livid, and brawny. This was most marked in the immediate vicinity of the gangrenous area. The œdematous condition had also extended higher up the leg. The gangrenous tissues were cut away and sulphur was applied in considerable quantity to the brawny œdematous adjacent tissues. An abundant hot boracic compress was applied all over the foot, and kept constantly hot and moist. A change rapidly took place in the character of the œdema, and within a few days all danger of the gangrene spreading had come to an end. Sulphur was applied frequently and any dead tissue was cut away. When the wound was quite clean a graft was applied.

You will readily gather from the character of the conditions which I have described that nothing short of amputation would have stayed the rapidly spreading septic gangrene in this man, whose vitality was enormously lowered by alcohol, if sulphur had not been used. The ordinary germicidal agents in common use were tried most thoroughly, and apparently without benefit.

A. L—, æt. 12, was admitted into Guy's Hospital under my care on July 8th, 1894, suffering from a large tubercular cavity which involved and had destroyed a portion of the wall of the rectum and internal sphincter, so that besides an abundant discharge of pus the child was quite unable to control its motions. The disease had begun twelve months before, apparently as an ischio-rectal abscess. The inner surface of the ischium was bared by the tubercular process, and was carious. Besides the large opening into the rectum there were several fistulous openings externally. The child was very feeble

and emaciated, and was rapidly losing flesh. The cavity was exposed as freely as possible, and was found to be very extensive, reaching high up into the pelvis. A large quantity of tubercular matter was removed, and the space was plugged with gauze saturated with sulphur, glycerine and sulphur being introduced at intervals subsequently. The improvement in the child's general and local condition was rapid and unmistakable. The cavity filled up quickly, and at the present moment the child is practically well, being able to control her motions and enjoying good health. This case showed quite as well as the others the rapid change in the general condition which is brought about by the removal of the bacillus of tubercle from the body, and the abrupt cessation of the absorption of any of its products.<sup>1</sup>

E. A—, æt. 20, had suffered from very destructive tubercular disease of the knee for more than six years, the joint having been disorganised and dislocated for a long time.

The joint was excised on October 13th, 1894, when, besides a very extensive tubercular infection of the synovial membrane and of the soft parts, the bones were found to be diseased through a considerable length. A very large sequestrum was removed from the femur. The sawn surfaces were retained in accurate apposition by means of silver wire, as, in spite of a very prolonged operation, owing to the very boggy condition of the surrounding soft parts, I feared that I had not completely removed the diseased structures. Gauze saturated with sterilised sulphur was packed into the space from which the tubercular material was removed, and left there for twenty-four hours.

A slough formed which gradually separated, the part being kept thoroughly dry and aseptic by the use of

<sup>1</sup> Some time after leaving the hospital a portion of the wound broke down again. This, in all probability, would not have happened could we have kept her long enough.

sterilised dressings. The inflammatory thickening subsided almost at once, and after seven days had elapsed the temperature never rose above  $99^{\circ}$ .

As far as I can speak from experience, I am certain that this case would not have progressed in the same satisfactory way if I had not made use of sulphur in the manner described.

E. W—, æt. 34, had suffered fourteen years ago from tubercular disease of both testes, which subsided after prolonged suppuration, and for nearly thirteen years from tubercular abscess in the perinæum, ischio-rectal fossa, and prostate.

These had for the last nine months communicated with the rectum on a level with the prostate, the mucous membrane of the rectum being deeply ulcerated about the aperture, through which urine escaped involuntarily.

In April, 1894, I had laid the cavities open as freely as possible, and had plugged with iodoform and gauze, with the result of rendering his condition decidedly worse, since my incisions rapidly became involved by the tubercular process.

The injection of sulphur and glycerine was commenced on September 4th, 1894, when the patient weighed 8 st. 10 lbs. He was shown at the Clinical Society on November 9th, when he weighed over 10 st. The rectal fistula had closed, and except for the escape of a few drops of urine occasionally from a minute perinæal fistula the patient was practically well.

Although only a fortnight has elapsed since he was last weighed, he now turns the scale at 10 st. 6 lbs., a gain of 1 st. 10 lbs. in two months and a half. No urine now discharges through the very minute orifice in the perinæum, and I expect this to close within a few days. The result obtained in this case was quite beyond my most sanguine expectation.

E. P—, æt. 50, was admitted into Guy's Hospital under my care on September 27th, 1894, suffering from a large

carbuncle on the left shoulder. It had begun to discharge externally, and was surrounded by an extensive area of thick brawny tissue.

The slough was removed the same day, when the cavity left measured at least an inch and a half in depth. It was plugged firmly with gauze and sulphur, and a hot compress was applied. The plug was removed at the end of thirty-six hours, after which the hot compresses were continued. Five days after the operation the edges of the surrounding skin were nearly level with the floor of the ulcer, from which the soft slough left by the action of the sulphur had almost completely separated. A graft was applied a few days later. Though I have scraped out a good many carbuncles, and have plugged the cavities left with iodoform and gauze, I have never before seen the same rapid subsidence of the thickened infiltrated edge and the early recovery of the healthy appearance of the floor that took place in this case.

I think I have related the details of a sufficient number of cases to indicate to you pretty clearly the action of sulphur and the various modes in which it may be used. From a consideration of them you will see that its range of application is a very wide and important one. I have availed myself of it largely for every infective process in which it could be applied locally. For instance, in tubercular disease of the bladder it is introduced as a traga-canth emulsion through a catheter; in the various conditions included under the very vague term "whitlow" I have derived immense advantage from its action, whether I have employed it in the form of a powder to fill a sloughy cavity in the subcutaneous tissue, a suppurating tendon sheath or joint, or an infective process running up beneath the annular ligament into the forearm. In these latter circumstances an emulsion distributes itself more rapidly and acts more effectively than the powder.

In lupus of the nasal cavities or of the mouth sulphur produces the same result as it does when applied in lupus

of the skin, though in the case of the mouth and throat it is obviously difficult to keep the sulphur in contact with the ulcerated surface. This difficulty can, however, be met by perseverance and ingenuity on the part of the surgeon.

For foul offensive ulcers, for some varieties of eczema, for the infective sores which occur about the head and buttocks of badly fed children, &c., it is equally valuable. When possible, a hot moist compress should be used as it stimulates the affected parts, and so produces more active chemical change.

As far as I know, sulphur has not been used in this manner before. On talking it over with an old friend of mine, he told me that he remembered seeing it dusted on poultices when he was first apprenticed to a surgeon.

Dr. de Rey Pailhade, a civil engineer of mines and also a doctor of medicine, has been experimenting on the medicinal action of sulphur, and has come to the conclusion that there is diffused through the animal kingdom a principle he calls philothion, which combines with sulphur at 40° C., producing sulphuretted hydrogen. For full information as to the evidence upon which this assumption is based I would refer to that author's elaborate works.<sup>1</sup>

Mr. Pakes is making a research into the several chemical changes which result from the introduction of sulphur into the human body, and hopes to lay the results before the Society at a future meeting.

I need hardly point out that as tubercle very rapidly relapses when the part appears quite healthy the patient must be kept under treatment for some considerable time after apparent recovery. The neglect of this precaution is well illustrated by the case of A. L.— (see foot-note, p. 63).

<sup>1</sup> 'Recherches expérimentales sur le philothion,' 8vo., Paris, 1892; 'Rôle du philothion dans l'absorption du soufre,' 8vo., Paris, 1892; 'Le philothion et le soufre,' 8vo., Toulouse, 1894; 'Rôle physiologique du philothion dans la respiration des tissus,' 8vo., Toulouse, 1891.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. vii, p. 25.)

# THE INFLUENCE OF HEREDITY IN PHTHISIS.

BY

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THE question of Heredity in Phthisis is one of great importance and of some difficulty. The importance of the subject cannot be doubted, for there is hardly a family in the country in which phthisis has not claimed a victim in one or more generations. If heredity be a potent cause of the disease, how few are the individuals who could hope to escape! But is the disease hereditary? The difficulties of deciding the question are numerous. Not only is the collection of the necessary *facts* far from easy from the want of knowledge or the want of accuracy on the part of patients in giving their family histories, but the *deductions* from these facts when obtained are by no means clear.

It is difficult also to approach the question with an unbiassed mind, in the face of the almost universal acceptance of the hereditary nature of consumption handed down from the past. The universality of the belief in the hereditary nature of consumption is easily explained when we know how frequently phthisis is found

attacking several members of the same family in successive generations; and anyone who wished to uphold the doctrine of heredity in phthisis would have no difficulty in amassing facts in support of his thesis. It is the apparent self-evidence of the matter which makes it difficult to conceive that there is any room for discussion. As Wilson Fox remarks, "the influence of this cause (heredity) in the production of phthisis is unquestioned." That phthisis is more common in some families than in others is not open to question; but the same is true of almost any disease, or indeed of any occurrence. If we were to investigate the statistics of scarlet fever or of malarial fever, we should find apparent support for a contention that these are hereditary; for undoubtedly we should find these diseases occurring repeatedly in some families, and never recorded in others. A family in which for generations the sons have served in the army and navy would possibly show malarial disease in some of its members for several generations; but this would not prove any hereditary transmission of malarial taint. The occurrence of ague for generations in families living in the Fens is no proof of the hereditary nature of malaria; nor is the frequency with which pediculi attack the children of successive generations of some poor families any evidence of heredity in this occurrence. In the above instances the mode of life, place of residence, and surrounding conditions are easily seen to exert so powerful an influence in determining the morbid condition as to furnish a complete explanation of its causation; and we at once agree that the greater incidence of these conditions on certain families is due to the members of them following the same profession, or living amidst similar surroundings. With phthisis, however, we are less able to see clearly the relative value of hereditary influence on the one hand, and of occupation and surroundings on the other. It is only within the present generation that the nature of pulmonary consumption has been determined, and the influence of



the surroundings and occupation of the individual on its causation more accurately studied.

Beliefs which have been held for ages are not easily relinquished, even when the contentions upon which they were founded have been disproved. It requires a more ample array of facts and more accurate reasoning to disprove an old theory than to successfully launch a new one. In the present case my inquiry has led me to consider the old theory of heredity in phthisis to be fallacious. I did not propose to myself, in preparing this paper, the task of trying to disprove the theory that phthisis is an hereditary disease. I set myself to analyse, as far as possible, the notes of 1000 cases, chiefly drawn from my hospital records; and to see how the facts, as I find them given in these ward notes, fit in with the theory of heredity.

In the foregoing preliminary remarks I have merely endeavoured—with the object of clearing the ground and counteracting prejudice in favour of the heredity theory—to urge that the question of hereditary influence is still open. I may at once state that, though I think my figures warrant a conclusion that direct heredity plays a less important part in the causation of phthisis than has sometimes been supposed, there appears to be no doubt that a *predisposition to become phthisical*—if exposed to tubercular infection—is often dependent upon inherited constitutional weakness.

Before proceeding further it will be well to define more exactly what we understand by heredity as a factor in disease causation.

(A) There is first *direct hereditary transmission* of a disease, the development in the offspring before birth of a morbid condition derived from the parent. The child is born suffering from the disease. This is true hereditary transmission, and is exemplified in congenital syphilis; it also appears in some inherited deformities, such as supernumerary fingers or toes. The disease is then properly called “congenital.” This kind of heredity is seen in

those cases, comparatively few in number, in which tuberculosis has been found in the young of animals *in utero*, or in the infant at birth.

(B) Then, secondly, the disease may be implanted in the body before birth, but does not manifest itself until some time after, *e. g.* infantile syphilis.

It is almost impossible to prove the occurrence of this mode of transmission for tuberculosis; for there are so many ways in which the causative bacillus may get access to the body of the infant after birth. It is, therefore, difficult to say whether a tuberculosis, showing itself some weeks after birth, is inherited (congenital) or acquired; in the majority of cases I should suspect the latter mode of origin.

In both the above categories a definite germ of disease is implanted; they differ thus essentially from the next two.

(c) Of these, the most common way in which heredity acts in disease causation is that there is transmitted a peculiar liability or susceptibility to develop a certain disease if the individual be exposed to the exciting cause of that particular disease; there is a diminished resisting power with regard to some special ailment. Here a predisposition is inherited, not the disease itself, and we may call this "direct hereditary predisposition." Examples of this form are numerous, as in gout and insanity. This susceptibility may be transmitted by parents who, though themselves inheriting the predisposition, have escaped the disease,—"*atavism*." It may be noted that this escape of the susceptible person shows that the inherited tendency may be combated and the disease itself avoided by avoiding the determining or exciting cause.

(D) In the last form the inherited susceptibility to disease may be general, not special, *i. e.* a liability to take any of a wide range of diseases to the exciting cause of which the person may subsequently be exposed. There is a diminution of resisting power against disease in

general. We may term this general inherited predisposition to disease, or delicacy of constitution.

Direct hereditary predisposition is what is now generally implied when consumption is said to be inherited.

As we are not now discussing tuberculosis in general, we may dismiss, for the purposes of this paper, the two first-mentioned forms of heredity, *i. e.* congenital diseases. Phthisis, or primary pulmonary tuberculosis, is essentially a disease of adult life, and it is difficult, with our present knowledge of the causative micro-organism of tuberculosis, to imagine the bacillus remaining latent in the body from birth to maturity.<sup>1</sup>

We have, then, to consider—

- (1) Direct hereditary predisposition to phthisis.
- (2) General inherited delicacy of constitution in relation to phthisis.

The first being a definite tendency to a particular disease, transmitted from parent to offspring, would be a true heredity, and as such is a matter of considerable importance. It behoves us, therefore, carefully to inquire whether it can be proved to exist for phthisis, and if so to what extent.

The second, though having an important bearing on the future health of the individual, hardly comes pro-

<sup>1</sup> Cases frequently occur which tend to show that the bacillus of tuberculosis may remain potentially active in the body for a long time without causing symptoms, and may yet eventually light up fatal disease. Encapsuled masses of tubercular disease, containing bacilli, may remain for considerable time in the lung without any apparent effect on the health, until in some way the bacilli escape and infect other parts of the lung, the disease then rapidly spreading. An example of the escape of bacilli from such an encapsuled mass was shown by me at the Pathological Society last session. Those cases in which the individuals in early life suffered from strumous glands—tuberculosis of the superficial cervical lymphatic glands—and then in later life develop pulmonary tuberculosis, may be examples of a similar event; though it is impossible to exclude the influence of a fresh infection of the lungs from without. The comparative immunity of the sufferers from strumous glands in childhood from subsequent phthisis is against the theory of auto-infection in these cases.

perly under the designation of an hereditary predisposition to phthisis, since it might equally plausibly be considered an hereditary tendency to scarlet fever or to yellow fever, or to almost any disease to which the individual might become exposed. This is in fact the essential point to which I wish to draw attention, and which I have here set myself to examine. Is the inherited predisposition, which is at present considered so important and so potent a factor in determining the incidence of consumption, a true heredity, or is it merely an illustration of the fact that those of delicate constitution, from whatever cause, are more liable to contract diseases to which they become exposed than those who are constitutionally robust?

Again we find it necessary to define terms somewhat more exactly. What do we understand by "delicate" and "robust" constitutions?

The breeders of animals know well how much of the usefulness and capacity for work depends upon the stock from which the animal springs. They recognise that only selected animals should be utilised for breeding purposes, and that these stud animals must be kept under certain conditions as to food, air, and exercise, in order that their offspring may turn out valuable. They endeavour to ensure that whatever peculiarities of shape, colour, or disposition the young animals may inherit from their parents, they shall have no blemish and no inherited liability to illness. The offspring of healthy parents who have been living in accordance with hygienic requirements will probably be themselves healthy, and if kept under similar hygienic conditions will in all probability grow up strong and capable of a full measure of work. Such animals, if exposed to unfavourable (unhygienic) conditions, such as extra strain of work or unhealthy surroundings, are less likely to break down, or will withstand the evil effects of these conditions for longer, than animals less carefully bred. They have "robust" constitutions — the strength which lies in

healthy tissues and healthy organs performing their functions naturally and harmoniously. Divergence from this desirable condition, if habitual, constitutes "delicacy," the ever-present liability to break down under a strain, or to become *invalid*.

Suppose the constitutionally robust animal to be afterwards placed under conditions unfavourable to health, his inherent strength may prevent a break-down. But although not suffering from any definite disease, the animal, whose tissues and organs are now unable perfectly to perform their several functions, would, in all probability, produce offspring similarly deficient in functional vigour—i. e. constitutionally delicate. A stage further in the downward course, and the animal would be incapable of producing offspring at all. This, then, illustrates the well-known fact that unhealthy offspring may come from parents who have no actual disease.

The same is true for human beings: if the parents are not strong and healthy, the children will be delicate in constitution, less able to bear the strain of work and anxiety, and more easy victims to disease than the children of perfectly healthy parents. The debility of the parents is not necessarily due to disease; it may be dependent upon unhygienic surroundings, yet the result is similar in the children—they have less power of resistance against disease. The Londoner of the fourth generation is said not to exist; and even if this statement oversteps the bound of literal accuracy, it is undoubtedly true that the conditions of life in the crowded slums of a large city tend to diminution of vitality in the individual. The children of parents thus debilitated, and themselves brought up in the same unhygienic surroundings, are weakly and delicate, and have small resisting power against disease, whether it be tuberculosis or some other malady. The particular disease which attacks them depends upon the liability to exposure to one or another of them. The debility of the parents may, on the other hand, be due to their being consumptive; and if this

cause a greater liability to the children becoming phthisical—if there be a special or specific hereditary predisposition—we should find the children of such parents phthisical in greater proportion than the children of parents whose debility arises from other causes. Let us then carefully examine our figures to see how far this is the case (see Table I). We find here that out of 1000 cases of phthisis, one or both parents were consumptive in 325, or 32·5 per cent. In 474 of these cases I have obtained a fuller account of the family, and of these 199 (nearly 42 per cent.) give a history of phthisis in the parents. In the families of the non-phthisical parents, 24·87 per cent. of the children became phthisical, against 33·71 per cent. of the children of the phthisical parents.<sup>1</sup> This shows a difference of only about 9 per cent.—a very small influence in favour of hereditary predisposition.

This additional 9 per cent. of phthisical children in those families in which the parents are consumptive might be explained by the increased exposure to the exciting cause, to which those living with consumptive parents are necessarily liable. Dr. Philip, of Edinburgh, in an analysis of 1000 cases of phthisis, put the percentage of cases in which the disease seemed to him to be due to direct infection at seven, or very nearly the whole proportion which, in the figures given above, represents the possible influence of hereditary predisposition.

It is argued that the parents may transmit this specific predisposition from their parents, without being themselves consumptive; so that to exclude the possibility of hereditary transmission in any case we should know the history of earlier generations. We might grant this, and yet take a very hopeful view of the effect of hereditary influence in phthisis. It seems probable that inherited peculiarities become weakened in each successive

<sup>1</sup> It is remarkable how closely the figures obtained by two different modes of inquiry approach to one another. The proportion of phthisical patients who give a history of phthisis in their parents is 32·5 per cent., whilst the proportion of the children of phthisical parents who became consumptive is 33·71 per cent.

generation, and tend to die out unless strengthened by intermarriage with a family in which the same peculiarity is strongly marked. If, therefore, the offspring of phthisical parents escape the disease, they transmit the predisposition with a diminished potency, and their children should be even more able to escape. I would lay stress upon this point, that however frequent we may find the hereditary influence to appear in statistics, it is not so strong as to cause alarm; though it may be strong enough to serve as a caution, and to indicate the necessity for extra care in avoiding the determining cause of the disease.

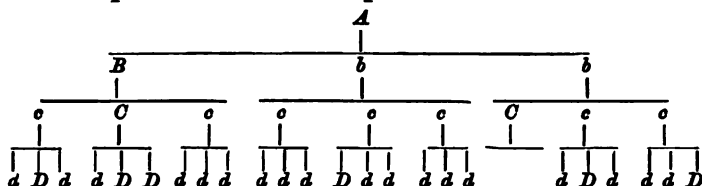
We will now proceed to a fuller examination of our figures, and see what we can glean from them. In the 1000 cases a history of phthisis in one or both parents was obtained in 325 cases, giving a proportion of 32·5 per cent. of the total number of cases; 604 were males, and of these 175 had a history of phthisis in the parents, *i. e.* 28·97 per cent. The 396 females gave a history of phthisis in the parents in 150 cases, or 37·87 per cent.

So far we see that in 1000 families in which one or more members of one generation became consumptive, one or both parents also had phthisis in about a third of the whole number. If we were to include uncles and aunts and grand-parents, the proportion of families in which phthisis was present in previous generations would be increased to about 35 to 40 per cent. This would seem to show a fairly strong hereditary influence, but on the other side we must put the not inconsiderable number of families in which phthisical individuals have had children and grandchildren free from the disease, and which necessarily do not appear in statistics taken from the records of hospitals for consumption. We are at once face to face with one great difficulty in attempting to decide the influence of heredity in consumption. To obtain reliable results we should commence with phthisical individuals, and obtain the history of their children and grandchildren, or their offspring for several generations;

and compare the result as regards the incidence of phthisis with that in the descendants of a similar number of individuals who were not consumptive. This would require much laborious searching of family records, or the cumulative results of observations continued through several generations. All statistics which commence with the phthisical patient, and trace the family history back through one or more generations, give results as regards heredity which are fallacious, and which place the proportion of hereditary cases too high.

In illustration of this statement let us take a supposititious family with a very bad phthisical history. We have seen that of the children of phthisical parents, only about one third become consumptive. In the following scheme capital letters indicate phthisical individuals, the small letters those who remain free.

*Scheme of supposititious family with bad phthisical history.*  
*Capital letters indicate phthisical individuals.*



To illustrate how the influence of heredity appears unduly great if we trace back from phthisical patients *D*, instead of forward from *A*.

Here we have phthisis in the parent *A* attacking one in three of the children, and showing in two out of nine individuals in the third generation, and six out of twenty-four in the fourth generation. If we start with the last generation we have six cases of phthisis (*D*) of which two show consumption in the parents, a proportionate heredity of 33·3 per cent.

If we trace back to grand-parents the proportion showing heredity is 50 per cent., and going back one more generation we find it to be 100 per cent. Even if we only go back one generation and take the collateral



branches (uncles and aunts) the hereditary influence appears as high as 88·3 per cent., that is five out of six consumptives (*D*) show phthisis in parents or uncles and aunts (*O*). If, however, we commence with the phthisical patient *A*, we find the influence of heredity represented by 33·3 percent. in the children, by only 25 per cent. in two generations, and by 25 per cent. in three generations, in spite of the large number of consumptives in the last generation.

We might, under unfavourable conditions of life, find quite as large a proportion of phthisical persons in three generations derived from a perfectly healthy stock.

Seeing, then, how fallacious will be the results obtained in the ordinary way, I have endeavoured to find out how far the children of phthisical parents are affected. I have not the details of the whole number of children in all the 1000 families, but only in about half of these, viz. 474. In these 474 families there were 275 in which neither of the parents was phthisical. These families comprised 1745 children (an average of 6·34 to each family); 935 were males and 787 females, and 23 who died in infancy, of whom the sex is not mentioned. If we divide these 23 proportionally between the sexes we should have 947 males and 798 females. Of the *male children* 111 died in infancy (11·87 per cent.), and of the remaining 836, 244 became phthisical, or 29·06 per cent. (see Table II).

Of the *female children* 82 died in infancy (10·41 per cent.), and of the remaining 716, 142 became phthisical, or 19·81 per cent. The proportion of phthisical invalids of both sexes is 24·87 per cent.

The above figures tend to show the influence of occupation in determining phthisis where there is no special predisposing cause in the family; for the males suffer out of all proportion to the females.

In 204 out of the above 275 cases I have noted the grand-parents and uncles and aunts of the patient, and in 179 there was no phthisis at all known in the family,

whilst in 25 cases there was phthisis in grand-parents or collaterals. I have endeavoured to find whether the proportion of individuals affected with phthisis is modified by this presence of consumption in the remote members of the family. The numbers are too small to be of much value, but my figures show that in the families where phthisis has been previously known, though the total incidence of phthisis is less than where no phthisis has been present (19·39 per cent. against 20·48 per cent.), the proportion of females attacked by phthisis is much greater in those families where previous examples of the disease are noted (males 16·25 per cent., females 22·85 per cent. as against males 27·78 per cent., females 18·69 per cent.) (Table III).

In 84 families (out of the 474) the father was phthisical.

These families comprised 511 children (an average of 6·08 to each family), of whom 267 were male and 244 female; 67 (or 13·11 per cent.) died in infancy, and of the remaining 444, 38 became phthisical, or 8·58 per cent.

In these families the incidence of phthisis is greater on the female children than on the males (35·18 per cent. as against 27·19 per cent.).

Delicacy of constitution is as potent a cause as the effect of occupation.

We see, also, the result of weakness in the father shown in the greater proportion of infantile deaths, especially amongst the male infants. The weakly sons having died off, the proportion of phthisis amongst the surviving male children is less than in the previous class, but the girls are more prone to phthisis than in families where there is no hereditary predisposition. We must remember, however, that the girls stay more at home than the sons, and are in closer contact with the phthisical parent, especially when the disease causes him to give up work and lie up.

In 82 families the mother was phthisical. These comprised 506 children (6·17 to each family), of whom 246

were male and 260 female. Fifty-six (or 11·06 per cent.) died in infancy, and of the remaining 450, 155 became phthisical, or 34·4 per cent.

Here, as in families where neither parent was phthisical, the male children suffer proportionally more than the female (39·43 per cent. against 29·95 per cent.). And a greater proportion of male children die in infancy (13·41 per cent. male, female 8·84 per cent.). The proportion of females affected is actually smaller than in the families where the father was phthisical. It might be urged that the male children inherit the mother's characteristics, whilst the female children are most influenced by the male parent. This is the reverse of what Dr. Reginald Thompson gathered from his figures. When the mother is delicate the proportion of male children is deficient, whilst those that are born are wanting in stamina, as shown by their liability to phthisis.

In 33 families both parents were phthisical; and in these there were 165 children (5·0 to each family). Eighteen died in infancy (10·9 per cent.), and of the remaining 147, 58 became phthisical, or 39·45 per cent.

The female children were only slightly more affected with phthisis than the males (38·8 per cent. of males to 40·0 per cent. of females). The infantile deaths are numerous amongst the male children, but few amongst the girls. In the survivors phthisis is common.

In the above families where phthisis is noted as having attacked one or both parents, it cannot be stated whether the disease was present before the birth of the patient. In the majority of cases it is probable that at the patient's birth the parent was non-phthisical.

To sum up the preceding remarks, we see that if we take 1000 cases of consumption, we get a history of phthisis in one or both parents in 325 cases, or 32·5 per cent.

We see also that in families of which one or both parents are phthisical, 33·71 per cent. of the children suffer from the disease; whereas in families with no consumption in the parents, 24·87 per cent. of the children become

phthisical. Phthisis in the mother results in a higher percentage of phthisis in the children than when the father alone is phthisical. This might be expected, without conceding any hereditary influence, from the more intimate relation between mother and children. When both parents are phthisical 39·45 per cent. of the children become consumptive.

It seems, then, that the influence of heredity may be represented by about 9 per cent.—the difference between the percentage of phthisis in the children of non-phthisical parents and those whose parents were consumptive. Even when both parents were phthisical the influence of heredity cannot be seen to result in more than fifteen children in every hundred becoming phthisical.

I have collected the following figures from various sources, so as to get the percentage in which phthisis occurred in the parents of consumptive patients from a sufficiently large number of cases.

		Cases.		Percentage heredity.		Number of cases in which the parents were phthisical.
Brompton (1st report)	...	1010	...	24	...	242
„ (2nd report)	...	4482	...	28	...	1255
Edward Smith	...	1000	...	21	...	210
Wilson Fox	...	884	...	38	...	127
Theodore Williams	...	1000	...	12	...	120
Philip...	...	1000	...	23	...	230
Müller	...	988	...	21·8	...	215
Koranyi	...	900	...	20	...	180
Wahl	...	745	...	26·4	...	197
My own cases	...	1000	...	32·5	...	325
Total		12,509	...	24·79	...	3101

We have here 12,509 cases, of which there was phthisis in one or both parents in 3101 instances, or 24·79 per cent. We may take it from the above figures that 25 per cent. is as a fair average for the extent of apparent heredity from parents.

Naturally when phthisical grand-parents and collaterals

are included we have a larger percentage of apparent heredity :

		Cases.		Percentage heredity.		Number of cases in which the parents were phthisical.
Brompton (2nd report)	...	4482	...	87	...	3899
Edward Smith	...	1000	...	56.2	...	562
Theodore Williams	...	1000	...	48	...	480
Wilson Fox	...	384	...	48	...	184
Cotton	...	1000	...	36	...	360
Müller	...	988	...	50.4	...	498
Bochdalek	...	3292	...	48	...	1580
Total	...	12,146	...	62.94	...	7563

The only comment I have to make on this second list is that the figures seem to me quite unreliable (especially as regards the Brompton report), for all who have much to do with London out-patients know how wide a term "consumption" is when employed by them in their family histories; it includes, amongst other diseases, bronchitis, asthma, anæmia, and alcoholism.

I have previously pointed out that we have only to carry the inquiry sufficiently far back in order to bring up the apparent proportion of heredity to 100 per cent.

We may note in the first of the above lists how closely the percentage of apparent heredity in Dr. Wilson Fox's cases coincides with my own; in both of these series the patients were drawn from the same class of people, chiefly London poor living under unhygienic conditions.<sup>1</sup>

It is also worthy of note that in the cases gathered from the London hospitals the percentage of apparent heredity is in excess of that for the total number of cases here shown. This might be taken to indicate that where the people are living under conditions favorable to the development of phthisis the apparent influence of heredity is greater than elsewhere. To my mind it tends rather to

<sup>1</sup> Having been house physician to Dr. Fox at University College Hospital, I am able to make this statement with confidence.—J. E. S.

show that mode of life and unhygienic surroundings, not inheritance, caused the disease in the children as in the parents.

The percentage of phthisis in the children of *non-phthisical* parents amongst my patients is as great as that representing the hereditary influence in the above list; this, again, tends towards the conclusion that the value of the influence of occupation and surroundings in the causation of phthisis is very much greater than that of any inherited tendency. The relative value, as a cause of phthisis, between heredity and the influence of occupation and surroundings is well shown in the difference between the percentage of cases where the parents were phthisical in Dr. Theodore Williams' 1000 cases from private practice and that of 1000 cases of hospital patients. Dr. Williams' cases give a proportion of apparent heredity of only 12 per cent. My hospital cases show a percentage of 32·5. Is not this almost sufficient in itself to negative the theory of direct heredity? For if hereditary influence were a potent factor in the causation of phthisis we should expect to find it showing nearly equally amongst the rich and the poor.

It has been stated that each sex has a tendency to transmit directly, fathers to sons, and mothers to daughters.

In the majority of cases, as far as I can gather, by which this statement has been supported, the figures are obtained by taking a single case in a family and finding out if either parent was consumptive. For example, a male patient presents himself with phthisis and states that his father was phthisical; without further inquiry this might lead to error, for he might have two sisters phthisical, and be the only consumptive son out of several in the family. To show that this way of taking the manner of transmission would lead to such an error, I have worked out this point in the way I have above alluded to for some of my cases. In 500 male patients the father was phthisical in forty-eight cases, the mother

in forty-six cases, and both parents in eighteen cases—showing transmission from father to son to be slightly in excess of cross-transmission, instead of as seen below.

By getting the history of all the children of a phthisical parent, as I have done in the present series of cases, we get a far more reliable result. It will be seen (Table II) that my figures are directly opposed to the theory of "direct" transmission.

The figures are—Father to sons	. 27·19 per cent.
Father to daughters	. 35·18 per cent.
Mother to sons	. 39·43 per cent.
Mother to daughters	29·95 per cent.

This shows a cross-transmission, as do also the figures arrived at by Dr. Wilson Fox from his 384 cases.

In order to see that my figures might not be misleading from dealing with an undue proportion of one sex amongst my cases, I have compared the proportion of males to females in my 1000 cases with the known proportion between the sexes amongst phthisical patients. I find my 1000 cases to consist of 604 males and 396 females. In 1000 cases recorded by Dr. Theodore Williams there were 625 males and 375 females.

The first Brompton report gives a proportion of 61 males and 39 females per cent.

The mortality from phthisis in London in 1880 gives for every 100 deaths, 57 males and 43 females.

Finally, amongst the 737 children in my tables who were phthisical, I find 418 males and 319 females, giving a proportion of 56·72 males and 43·28 females per cent. The proportion between the sexes in the 1000 cases dealt with in this paper is, therefore, about the relative proportion in which the two sexes are attacked with phthisis.

Of the 474 cases from which my tables as to children are drawn up, 309 were male patients and 165 female, a proportion of 65·19 males and 34·81 females per cent., or, again, not a long way from the proper relative proportion between the sexes.

## CONCLUSION.

The analysis of these cases warrants the opinion that the hereditary predisposition in the case of phthisis is not a direct predisposition to this malady, but merely the general predisposition to disease which belongs to the offspring of weakly parents; a predisposition which the children of phthisical parents have in common with those of parents whose health is lowered by other conditions. The families of consumptives have, however, the disadvantage, not shared by the delicate children of non-phthisical parents, of living in constant association with a source of infection. They have a general predisposition to take any disease to the infection of which they may be exposed, and being constantly exposed to the infection of tuberculosis they are prone to become consumptive. This circumstance may well account for these families showing an incidence of phthisis only 9 per cent. in excess of the incidence of the disease amongst the children of non-phthisical but weakly parents.

I believe that the present criticism of the influence of heredity by the examination of the total number of children of one generation in a phthisical family deals with a larger number of cases than has as yet been published.

The results of the examination of the figures may not go very far in deciding any of the various questions which surround or are included in the theory of inheritance in phthisis. The figures themselves may, however, be of some interest and value even to those who cannot agree with the deductions I have drawn from them.



## STATISTICS.

Of 1000 phthisical patients 325 gave a history of phthisis in one or both parents. Apparent heredity = 32·5 per cent. Male patients 604 ; apparent heredity 28·97. Female patients 396 ; apparent heredity 37·87.

TABLE I.—*Showing the incidence of phthisis in the children of non-phthisical and of phthisical parents respectively.*

474 CASES.	CHILDREN.		DIED IN INFANCY.		PHTHISICAL.	
	Total number.	Proportion to each family.	Total number.	Per cent. of whole.	Total number.	Per cent. of children, omitting infantile deaths.
A. 275 families. With <i>no phthisis</i> in parents }	1745	6·34	193	11·06	386	24·87
B. 84 families. <i>Fathers phthisical</i> }	511	6·08	67	13·11	138	31·8
C. 82 families. <i>Mothers phthisical</i> }	506	6·17	56	11·06	155	34·4
D. 33 families. <i>Both parents phthisical</i> }	165	5·0	18	10·9	58	39·45
199 families. Phthisis in one or both parents (Summary of B, C, D) }	1182	5·93	141	11·92	351	33·71

TABLE II.

APPARENT HEREDITY IN 474 CASES.	SEX OF CHILDREN.	CHILDREN.		DIED IN INFANCY.		PHTHISICAL.	
		Total number.	Proportion to each family.	Total number.	Percentage of whole.	Total number.	Percentage of children, omitting infantile deaths.
A. Nil. 275 families	M.	947	3.44	111	11.87	244	29.06
	F.	798	2.90	82	10.41	142	19.81
B. Paternal. 84 families	M.	267	3.05	39	14.60	62	27.19
	F.	204	2.90	28	33.3	76	35.18
C. Maternal. 82 families	M.	246	3.00	33	13.41	84	39.43
	F.	260	3.17	23	28.04	71	29.95
D. Double. 33 families	M.	84	2.54	12	14.28	28	38.8
	F.	81	2.45	6	7.47	30	40.0
Summary of B, C, D. 199 families	M.	597	3.00	84	14.07	174	33.91
	F.	585	2.93	57	9.74	177	33.52

TABLE III.—Showing a further analysis of 204 (from Cases A in the previous table) in which the history of grandparents, uncles, and aunts could be determined.

204 CASES WITH NO PHTHISIS IN THE PARENTS.	SEX OF CHILDREN.	CHILDREN.		DIED IN INFANCY.		PHTHISICAL.	
		Total number.	Proportion to each family.	Total number.	Percentage of whole.	Total number.	Percentage of whole, omitting infantile deaths.
179 cases with no phthisis previously known in the family	M.	581	3.24	70	12.04	142	27.78
	F.	522	2.91	62	11.97	86	16.69
25 cases, phthisis in grandparents or collaterals	M.	84	3.36	4	4.76	13	16.25
	F.	89	3.56	4	4.49	19	22.35

*Table showing the proportion of phthisical children to 100 non-phthisical in 80 cases from Dr. R. Thompson's table of heredity (p. 45 of his book), compared with a similar proportion in 199 cases of my own with phthisis in one or both parents. (See totals in the first two tables.)*

		Phthisical.		Non-phthisical.	
Dr. R. Thompson.	{ Males . . . . .	116.1	100		
	{ Females . . . . .	137.14	100		
My own cases	{ Males . . . . .	51.32	100		
	{ Including all infantile deaths	76.10	100		
	{ Females . . . . .	50.42	100		
	{ Including all infantile deaths	66.6	100		

*Table showing the number of males and females who developed phthisis out of 474 families, in 199 of which heredity was suspected.<sup>1</sup>*

Heredity.		Phthisical.		Died in childhood.		Non-phthisical.	
Nil 275 . .	{ Males . . . . .	947	244	111	592		
	{ Females . . . . .	798	142	82	574		
Paternal, 84 . .	{ Males . . . . .	267	62	39	166		
	{ Females . . . . .	244	76	28	140		
Maternal, 82 . .	{ Males . . . . .	246	84	38	129		
	{ Females . . . . .	260	71	28	166		
Double, 33 . .	{ Males . . . . .	84	28	12	44		
	{ Females . . . . .	81	30	6	45		
Total of 199 hereditary cases	{ Males . . . . .	597	174	84	339		
	{ Females . . . . .	585	177	67	351		

*Table showing the number of males and females who developed phthisis out of 80 families in whom heredity was suspected (from Dr. R. Thompson).*

Heredity.		Phthisical.		Died in childhood.		Non-phthisical.	
Paternal, 24 . .	{ Males . . . . .	39	20	2	17		
	{ Females . . . . .	54	30	5	19		
Maternal, 30 . .	{ Males . . . . .	102	38	9	55		
	{ Females . . . . .	78	34	4	40		
Double, 14 . .	{ Males . . . . .	34	17	9	8		
	{ Females . . . . .	30	17	5	8		
Atavism, 12 . .	{ Males . . . . .	28	23	1	4		
	{ Females . . . . .	20	15	2	3		
Total (80 hereditary cases)	{ Males . . . . .	203	98	21	84		
	{ Females . . . . .	182	96	16	70		

<sup>1</sup> For comparison with Dr. R. Thompson's Table III, p. 45 of his work.

<sup>2</sup> Table III of Dr. R. E. Thompson (p. 45).

(For report of the discussion on this paper, see ' Proceedings of the  
Royal Medical and Chirurgical Society,' Third Series, vol. vii, p. 29.)

ILLUSTRATIONS  
OF  
SOME MODES OF DEATH AFTER  
OVARİOTOMY

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IN the study of surgical procedures it is sometimes advantageous to discuss the methods of operating, and the principles which should guide our treatment, whilst at other times it is more instructive to relate cases, and to endeavour to show how success has been obtained, or how failure might have been avoided. Discussions on the methods of ovariectomy have been so frequent, and I have expressed my own views regarding them so fully, that I now propose to have recourse to the second plan.

I need not, however, on the present occasion report cases in illustration of the successful application of the views on the condition and management of the intestine after abdominal section which I first published in 1887, and which have the honour of being recorded in our Society's 'Transactions.'<sup>1</sup> In that year I drew attention to the fact that symptoms of obstruction and paralysis of the bowel had often been mistaken for those of peritonitis after operations on the abdomen, and notably that the cases of peritonitis which Mr. Lawson Tait professed to

<sup>1</sup> Vol. lxxi.

cure by purgatives were not cases of peritonitis at all. I mention Mr. Tait because he has declared that he has been "grossly misrepresented and misunderstood" in this matter, and that he "has never said that the purgative treatment will cure peritonitis."<sup>1</sup> As this statement has recently been brought prominently forward before the Medical Society,<sup>2</sup> I take the opportunity of pointing out that the exact words of Mr. Tait against which I argued were these:—"Now we beat the peritonitis. On the slightest indication of peritonitis after an ovariectomy we give a rapidly acting purgative, it matters not what, the patient's bowels are moved, and the peritonitis disappears."<sup>3</sup> My contention was<sup>4</sup> that the symptoms here referred to by Mr. Tait as indications of peritonitis were not due to this cause, that peritonitis did not exist in these cases, and consequently that peritonitis could not disappear,—a view with which Mr. Tait seems now to agree so fully that he has forgotten that he ever held any other.

I could give numerous cases showing success following attention to this matter, but there is abundant evidence in recent literature that the condition and the management of the intestines after abdominal section are now receiving ample attention on the lines which I suggested, and that a recognition of the importance of this matter is leading to a very marked improvement in the success of abdominal surgery.

Under these circumstances, and remembering the adage that we learn more from our failures than from our successes, I propose to relate in this paper the histories of all my cases in which death has been caused by operations for the removal of new growths of the ovary. But before doing so I should like to allude to certain cases in

<sup>1</sup> 'Brit. Med. Journ.,' 1892, vol. ii, p. 1050.

<sup>2</sup> "Lettsomian Lectures," 'Med. Soc. Trans.,' 1894, p. 178.

<sup>3</sup> 'Brit. Med. Journ.,' 1886, vol. i, p. 921.

<sup>4</sup> "The Condition and Management of the Intestine after Abdominal Section," 'Med.-Chir. Trans.,' vol. lxxi.

which I refused to operate on patients who, I believe, had tumours developing primarily in ovarian tissue. In publishing records of ovariectomy, surgeons not infrequently assert that they have made no selection of their cases for operation,—a statement that always surprises me exceedingly. It is a fact that some ovarian tumours are so malignant in their nature that before they are discovered they have infiltrated the tissues of the broad ligament in a manner that renders their successful removal impossible, so that no wise surgeon would make the attempt. It would really be a matter of extraordinary good fortune if an operator of any experience had never been called upon to refuse a case as malignant. If, however, he has declined to operate on account of this condition there is a question of diagnosis, and there may be room for a difference of opinion, because, without doubt, many cases of ovarian growth which it is difficult and dangerous to take away may simulate those cases having tumours of an irremovable malignant character. Thus mistakes sometimes occur, and I have to record two, in cases No. 3 and No. 6, and a third by another operator in my second fatal case. I have also notes of three cases in which I diagnosed ovarian tumours that could be removed, and I wished to perform exploratory operations, but in which my opinion was overruled. In one of these cases an ovariectomy was successfully performed by another surgeon. In the second case an ovarian tumour was found after death, but whether it could have been successfully removed is uncertain. In the third case there was neither operation nor post-mortem examination.

I have declined to operate in two cases of large nodular fixed tumours which probably originated in ovarian tissue. In both cases the peritoneal sac was distended with free fluid, and there was double pleuritic effusion, whilst in one of them the growth was fungating into the vagina at the site of a tapping puncture that had been made some time before I saw the patient. In a third patient on whom I refused to operate there was a cystic abdominal

growth, apparently ovarian, with solid deposit in the parietes, enlargement of the inguinal glands to the size of walnuts, infiltration of the left broad ligament, and complete fixation of the uterus. In a fourth case I successfully removed an ovarian tumour from a woman aged thirty-six years, on March 17th, 1892, and found no reason for taking away her second ovary. Within a year she was nearly as big as before the operation, but she did not consult her medical attendant, Dr. Alexander, of Epsom, till she got an extensive catarrhal pneumonia of both bases. I saw her at this time, and diagnosed another ovarian tumour, but I did not think there was any possibility of successfully removing the growth unless the condition of the lungs improved. The tumour was evidently, as the first had been, very multilocular, with no cysts of any great size, so that there was no advantage to be expected from tapping. The lungs did not improve, and the patient died on April 6th, 1893. Her tumour would have been removable with every prospect of success if advice had been sought sooner. In another case I removed a papillomatous tumour of the right ovary containing much solid matter, on May 12th, 1891. The second ovary seemed unaffected, but two years later, on May 4th, 1893, the patient had a tumour about the size of a cocoa-nut implicating the uterus. I declined to operate because the patient was over sixty years of age and very feeble, and I did not think the tumour could be successfully removed, even if she had been young and strong. She died in July of the same year. As far as my recollection goes, aided by my notes, these are the only cases of ovarian tumour in which I have refused to operate.

In dealing with cases of doubtful nature and unusual difficulty, I endeavour, in the first place, to determine whether there is any possibility of successfully removing the tumour by operation. If this seems at all feasible, I explain clearly to the patient the position in which she is placed, the danger that death may be an immediate con-



sequence of the operation, and the certainty of a fatal issue unless the tumour is removed. If the patient then wishes me to operate, I consider it my duty to undertake the responsibility of doing so even if the chances of success appear very small.

My first fatal case was that of a single woman, twenty-three years of age, placed under my care by Mr. Knowsley Thornton and Mr. Willans of Much Hadham. About three months before I saw her she had been suddenly seized with pain, vomiting, and an unnatural discharge from the bowels. The abdomen at the same time diminished in size, a considerable enlargement having taken place without being observed. Mr. Willans was not immediately called in, and when he saw the patient he at once arranged for her admission to the Samaritan Free Hospital, where she was received on March 7th, 1891. She was then extremely emaciated, her abdomen was greatly distended by flatulence, the pulse was rapid, and the temperature varied between  $100^{\circ}$  and  $103.4^{\circ}$ ,<sup>1</sup> but the discharge from the bowel had ceased. After eighteen days' treatment the flatulent distension had almost gone, and a tender tumour was discovered filling the pelvis behind the uterus and reaching to about two inches above the pubes. The temperature now ranged below  $101^{\circ}$ .

On March 25th, 1891, I removed through a median incision a cystoma of the right ovary containing about two and a half pints of extremely offensive dermoid material. The tumour was extensively adherent to surrounding structures, and at one point was so closely connected with the sigmoid flexure that I had to leave a portion of the cyst wall on the bowel. I made a second opening in the left groin, where this part of the bowel lay naturally against the anterior abdominal wall, and fixed the remaining piece of cyst in this opening outside the peritoneum, so that any discharges from it could escape freely. I

<sup>1</sup> All the temperatures recorded in this paper are Fahrenheit and were taken in the vagina.

drained the peritoneal cavity through the median incision, taking great care to keep apart the dressings and the discharges of the two wounds. When put to bed the patient was very weak. The temperature quickly rose to  $101.8^{\circ}$ , and then tended downwards. The pulse rose to 140 and continued fast. In other respects the condition was not unsatisfactory for the first two days; flatus passed freely from the rectum, and on the second day the bowels moved spontaneously. The discharge from the wounds was scanty and odourless. Early in the morning of the third day flatus did not escape at all freely when the rectal tube was inserted, and there were colicky pains on the right side. In the afternoon of this day the groin wound became very offensive, the temperature was  $101.4^{\circ}$ , and the pulse 144. In the evening the bowels again moved spontaneously, and the temperature fell to  $100^{\circ}$ . On the fourth morning the temperature was  $100.8^{\circ}$ , the pulse was 130, and there was occasional vomiting with slight distension of the abdomen, which latter diminished after the bowels were again moved by three small (3j) doses of sulphate of magnesia. After this very little flatus escaped from the bowel. Towards evening the distension increased, vomiting was frequent, the urine became scanty, the temperature rose to  $103.8^{\circ}$ , and the patient died very early on the fifth day after the operation. The discharge from the drainage-tube was clear, scanty, and odourless to the last.

At the autopsy, which Mr. Stephen Paget kindly made for me, there was very little fluid in the pelvis, and no sign of general peritonitis. The small intestine was adherent, near its lower end, to the stump of the pedicle on the right broad ligament. The bowel was acutely bent, and completely occluded at the point of adhesion, but otherwise healthy. Suppurative peritonitis had spread from the groin wound, but the neighbouring coils had become adherent, so that the inflammatory action was strictly localised. It was found to be impossible to separate the piece of cyst wall from the bowel.

In this case the occlusion of the intestine caused by its adhesion to the broad ligament, unless it had been relieved by operation, or by some chance alteration in the position of the adherent gut, would certainly have brought about the death of the patient under any circumstances. I have recorded a case<sup>1</sup> in which, in a strong healthy woman, an exactly similar condition developed, and was the only abnormality found after death. Had it not been for this accident, I am of opinion that a successful issue would have taken place. It is quite clear that the septic extension from the wound in the left groin did not begin till after the signs of obstruction became obvious. I have shown in other publications<sup>2</sup> that obstruction of the intestines has a most pernicious influence on any co-existing inflammatory condition ; and, on the other hand, a piece of cyst wall fixed as in this case outside the peritoneum, without tension, might fairly have been expected to give rise to no trouble if there had been no further complication.

The second fatal case was that of a widow, forty-six years of age, whose abdomen was tapped about fourteen times between November, 1880, and January, 1892. In the spring of 1891 a tumour was discovered, and the obstetric physician to one of our large hospitals opened the abdomen, but did not attempt to remove the growth. In January, 1892, the patient came to the Samaritan Free Hospital, and consulted Dr. Rutherford, who was strongly of opinion that another operation ought to be performed, and asked me to do it, at the same time informing me that a well-known abdominal surgeon had declined to undertake the case. I admitted the patient, and after removing by aspiration twenty-six pints of clear amber-coloured fluid from the abdominal cavity, I

<sup>1</sup> "On some Complicated Cases of Abdominal Section," 'Lancet,' July 18th, 1891.

<sup>2</sup> "The Physiology of Death from Traumatic Fever." "The Condition and Management of the Intestine after Abdominal Section," 'Med.-Chir. Trans.,' vol. lxxi.

easily defined a tumour rising to about two inches above the navel. It surrounded and was firmly fixed to the uterus, but the mass moved very slightly in the pelvis, which it nearly filled. The sound passed two and a half inches through the os uteri towards the middle of the growth. In answer to my inquiries, the surgeon who had done the exploratory operation kindly wrote that he had "performed abdominal section, but could do nothing, as there was extensive malignant disease (probably papilloma of ovary), and involving omentum and intestine." The general health of the patient was, however, still good when she came into my care about a year later; the tumour seemed to me removable, and of course it is known that ovarian papilloma is not necessarily malignant. I therefore asked Mr. Knowsley Thornton to see the patient in consultation, and with his approval I agreed to her urgent request that I should attempt to take away the growth.

I operated on March 14th, 1892, when the abdomen was again moderately distended. I had ordered that the urine was to be drawn off before the operation. This had been done without difficulty before the patient was tapped, but now two nurses tried and failed to pass a catheter. They decided to say nothing about the matter, and the consequence was that I cut into the enormously distended bladder,—an accident of which I have seen and heard so much, and which may so easily happen, that I have always been particularly anxious and careful to avoid it. In this instance I thought the bladder was quite safe, for I began by making a small incision near the umbilicus. Having emptied the bladder and closed the aperture with forceps, I opened the peritoneal cavity, and when a large quantity of fluid had escaped, a multilocular cystic tumour, almost completely covered by fungating papillomatous growth, was seen filling the pelvis and rising above the level of the umbilicus, adherent everywhere except on its anterior aspect, where it had been in contact with the free fluid. Its size having been reduced by tapping the

larger cysts, the separation of adhesions was begun, and it is to be noted that the adherent papilloma came completely away, leaving no sign of infiltration or extension into neighbouring parts. The growth filled Douglas's pouch and the space in front of the uterus, surrounding also the right broad ligament from which it grew, and its base was so solid that there was great difficulty in getting at the pedicle. By freely breaking up the tumour and tearing off large masses of papilloma, I was, however, able to expose and tie the broad ligament. These proceedings gave rise to much hæmorrhage from the tumour itself, and from numerous bleeding points of adhesion. When the tumour was removed and the bleeding was arrested I found that the broad ligament had not been divided sufficiently low down to permit of the complete removal of the neoplasm. I therefore transfixed and ligatured afresh beyond the disease, and owing to the shortness of the pedicle I had to tie the knots very tightly to avoid the danger of slipping of a ligature. A small pedunculate growth was removed from the top of the left broad ligament near the uterus. The left ovary was not enlarged, but it and the Fallopian tube were bound down by old adhesions. I now observed a condition of intestines which I have not seen in any other case. There was no small gut in the pelvis, nor in front of the spinal column. The intestines were all tucked away in the upper part of the abdomen and in the loins, and were fixed thus by firm adhesions of the mesentery and of the gut itself. The only free portion of the bowel was the sigmoid flexure, which had been attached to the tumour, and which now hung down very awkwardly in Douglas's pouch. A drainage-tube was placed in the pelvis and the incision was closed, the sutures being arranged over the bladder wound so as to avoid all possibility of escape of urine into the peritoneal cavity. As a further precaution, a Skene-Goodman catheter was placed in the urethra, the bladder was drained for three weeks, and it never gave any trouble.

The after history of this patient was in many respects similar to that of the first case, only it was prolonged for thirty-five days. She quickly recovered from the operation, and her condition gave no cause for anxiety during the first week, the highest temperature and pulse being  $101.8^{\circ}$  and 116 respectively. Urine was secreted abundantly, and soon became clear. Flatus passed downwards early, and there was no distension. The drainage-tube discharged between six and seven ounces of fluid in the twenty-four hours. A frequent cough seemed to be causing the tube to irritate the peritoneum, and I therefore removed it on the fourth day. On the sixth day the cough was much better, and expectoration was easier; the temperature was  $99^{\circ}$ , and the pulse 84 in the morning. During the second week the bowels were opened daily, or every second day, by enema or by laxative medicine. There was now, however, some distension in the lower abdomen, the edges of the wound became red, and the temperature varied between  $99.6^{\circ}$  and  $101.6^{\circ}$ , the pulse being between 86 and 100. The wound had healed by first intention, except superficially where the skin edges had not been exactly apposed, especially at the part where the drainage-tube had been fixed and over the bladder opening. On the sixteenth day the wound was completely healed and free from all signs of irritation. During the night between the sixteenth and seventeenth days no flatus passed from the anus, the patient suffered much pain, her abdomen became greatly distended, the temperature rose to  $108^{\circ}$ , and the pulse to 116. A turpentine enema caused a slight movement of the bowels and a free passage of flatus, with great relief of pain and diminution of the distension. The temperature fell by the following evening to  $100.2^{\circ}$ , and the pulse to 96. From this time there was frequent difficulty from retention of flatus, and the abdomen gradually increased in size, but the temperature was only once recorded above  $101.2^{\circ}$  for a few hours, on the twenty-fifth day, when it rose to  $108^{\circ}$  in the vagina. On the twenty-fourth day vomiting set in, and was at first very occasional, but

became more frequent later. On the thirty-third day the lower abdomen to about two inches above the umbilicus had become very hard and greatly enlarged, and as the wound had stretched it had again become red and irritable. Douglas's pouch was also distended and hard, and the rectum was found to be on the right side of the pelvis. It was obvious that the patient would soon die if left alone, but as she had endured so much, and still had considerable strength, I determined to seek for and attempt to relieve the obstruction which seemed to exist. The patient, who since the operation had always been quite sensible, told me to do whatever I thought best. Sir Spencer Wells saw her with me, and agreed with my view of the case. I therefore reopened the abdomen on the thirty-fourth day after the first operation, and found that the distension was due to an enormously enlarged and convoluted sigmoid flexure, the coils of which were matted together and adherent to everything they touched. The portion of the right broad ligament which was beyond the ligatures had sloughed, and was represented by a greyish stringy substance which lay in a cavity that it seemed exactly to fit. The ligatures were with it, and appeared as fresh as when tied. There was about an ounce of serous fluid in the upper part of the abdomen, but otherwise the peritonitis was altogether adhesive. The small intestines were exactly as at the first operation. I freed the adhesions of the sigmoid flexure completely, and attempted to release the small intestine, but with little effect. The peritoneal cavity was washed with boiled water and drained.

The patient was put to bed with a temperature of  $101.8^{\circ}$  in the vagina, and a pulse of 120, and she quickly recovered from the anæsthetic. Two ounces of clear urine were drawn off by catheter three hours after the operation, and one ounce after three hours again. The patient was quite conscious and sensible for a few hours, but later she became violently delirious, and she was still so strong that it required considerable exertion on the

part of two nurses to keep her in bed. The temperature gradually fell to  $97.6^{\circ}$ , the pulse rose, and death followed fourteen hours after the second operation. No sign of malignant disease was found at the *sectio cadaveris*.

The adhesions of the intestine in this case seemed to me to be due to inflammation caused by the irritating fluid to which the peritoneum had been exposed so long, owing to the delay in removing the tumour that had unfortunately taken place. The greater part of the intestinal tract could not expand, and the sigmoid flexure was therefore left without the natural support of the small intestines. When the patient began to take food by the mouth, the sigmoid flexure became distended, and being adherent in the pelvis, became convoluted, and gave rise to an incomplete obstruction.

It is, of course, possible to argue that the sloughing of the distal portion of the pedicle was the cause of the adverse symptoms. It is, however, most unlikely that the strangulated portion of the broad ligament would live for a time and then die. All the conditions were favorable to its complete recovery if it survived the first few days. In a very short time the ligatures would yield a little, the pedicle itself would shrink, and adhesions would form. I had been obliged, however, in this case to cut the pedicle very short, and tie it as tightly as I could, in order to get the growth away and avoid the possibility of slipping of the ligature. There is, therefore, a strong presumption that the ligature was too tight, and that the slough had existed from immediately after the operation. But the first signs of real danger did not appear till sixteen days later, whilst there was no sign at all for nine days. Moreover at the second operation the slough had a stringy appearance, as if its softer parts had already been absorbed. There is no doubt that aseptic organic matter in the peritoneal cavity may be absorbed, and absorption, if it ever occurs, might have been expected to take place in a patient possessed of the remarkable physical and healing powers exhibited in the case under consideration. It



seems clear that the slough was not septic, for, if it had been, an extensive pus formation must have taken place. For these reasons it seems to me that if the obstruction from extreme dilatation of the sigmoid flexure had not occurred, the condition of the pedicle would never have been known or suspected.

The unfortunate accident to the bladder cannot be considered in any way the cause, or even a contributing cause, of the fatal issue in this case.

The third case was sent to me by Dr. Searson, of St. Peter's Park, in July, 1892. The patient was thirty-one years of age and had been married only a few months.

I diagnosed a small ovarian tumour, not rising out of the pelvis, and closely attached to the uterus as if by a short pedicle, or growing in the broad ligament. As the Samaritan Hospital was about to close for the annual cleaning, I told the patient and Dr. Searson that she must wait till October or seek advice elsewhere. It was decided to wait, and the more readily because the patient's husband—a sailor—was expected home about October. I did not think the delay would hurt the patient, but the development of the tumour was extremely rapid. When I saw her on admission to hospital, a tense, smooth, tender growth greatly distended the abdomen, which was dull on percussion as high as the umbilicus, the intestines being pushed upwards and forwards, so that the limits of the tumour above were not definable. Fluctuation was distinct over the whole of the dull area and to some distance beyond it. The uterus was of normal size, was dragged or pushed up into the lower abdomen, and was easily felt through the abdominal wall immediately above the pubes, being held prominently forward by the new growth. The pelvis was distended by a large, smooth, tender mass, in which fluctuation was distinctly felt on tapping the abdominal wall. The pulse varied from 120 to 144. The temperature was usually subnormal in the mornings and from

100° to 101° in the evenings. The patient was otherwise healthy, but extremely emaciated and feeble. I thought the tumour was an ovarian cyst, probably extensively adherent. After consultation with my colleagues it was decided that the best chance of saving the patient's life was given by removing the tumour at once. If this proved impossible I thought I could at least give relief by emptying and draining the cyst. When I opened the abdomen I found a condition that had not been suggested by anyone who had examined the patient. The tumour was a soft solid, adherent to the abdominal wall. As there was absolutely no hope for the patient if I stopped the operation at once, and there might be some possibility of success if the adhesions happened to be limited to the anterior wall of the abdomen, I enlarged the wound and tried to get the tumour out. It had, however, no more consistence than a fresh blood-clot; its capsule was only very slightly firmer in texture than the bulk of the growth, and it was universally adherent, the adhesions being stronger than the substance of the tumour, so that when I attempted to separate the growth, pieces of it remained sticking to the bowels and abdominal wall. The tumour filled the pelvis and rose above the costal margins. It grew from the right ovary, and its base was of slightly firmer texture than the other parts of it. I tied the broad ligament below the growth and divided it beyond the ligature. Some masses of omentum were also removed, the chief bleeding points were tied, and large quantities of the growth were scooped out. I then closed the wound, knowing that I had only removed a portion of the tumour. The patient died under the shock of the operation.

At the autopsy much new growth was found in the pelvis and sticking to the omentum and intestines, which were matted together by adhesions. The mesenteric glands were, "with very few exceptions, not enlarged," and there were no secondary growths in any of the viscera. The left ovary contained two cysts, one the

size of a walnut, the other much smaller, being a dermoid cyst. The rest of the body was healthy, but very anæmic.

I do not know how the condition which existed in this case is to be diagnosed. The use of an aspirating needle would certainly not have revealed the solid nature of the tumour. The extreme rapidity of growth and the corresponding debility were marked features of the case, but could scarcely be called pathognomonic. Of course if a correct diagnosis had been made, no operation would have been performed, but the physical signs exactly simulated those of an adherent cyst.

The fourth case was that of a woman thirty-three years of age, who consulted Mr. Thornton in 1886, on account of a fibroma of the uterus reaching nearly as high as the umbilicus. An operation was not then considered necessary, and the patient was fairly comfortable till March, 1893, when she again sought advice because the tumour was rapidly increasing in size, and Mr. Thornton sent her into the Samaritan Free Hospital under my care. She had a large tumour extending into and expanding the cervix uteri, which was low in the pelvis. The growth reached four inches above the umbilicus, and fluctuation was distinct in the upper part, but just above the pubes and in the left groin the tumour seemed solid, and was very tender. The other organs of the body were all healthy, but anæmia was extreme. An ovarian tumour complicating the fibroma was diagnosed by Mr. Thornton.

I opened the abdomen on May 1st, 1893. The cystic and solid parts of the tumour formed one mass, the cyst being a tumour of the right ovary, inseparable from a uterine fibroid tumour over a large extent of their contiguous surfaces. The sigmoid flexure and cæcum were raised high up on the back of the enlarged womb, and on the left side there was another ovarian cystoma, the size of a small cocoa-nut. After a little enucleation on the right side both broad ligaments were tied and divided below the ovarian tumours. I then made an incision through the

peritoneum above the sigmoid flexure and cæcum behind, and above the bladder in front, so as to enable me to push these organs and the tied proximal portions of the broad ligaments downwards. I was thus able to get at and enucleate the base of the fibroid which extended very deeply into the left broad ligament. The iliac artery and vein on that side were exposed for some inches, and numerous vessels bled freely, but the cavity was stuffed firmly with sponges which arrested the hæmorrhage until I got the wire of a serre-nœud tightly secured round the base of the tumour, and cut it away, leaving little if any of the body of the uterus below the wire. The sponges were then removed, and the hæmorrhage was stopped, but the bleeding points had to be secured very carefully, for many of them were close to the iliacs, and the exact situation of the ureter was not discovered. The pedicle was fixed outside the abdomen in the lower angle of the wound, and the incision was closed with silk sutures.

The patient at first gave little or no cause for anxiety. The highest temperature in the first week was  $102^{\circ}$  F. on the night following the operation. The pulse was between 80 and 90, but very excitable, running up immediately to about 120 if the patient made the slightest exertion or saw a stranger. Flatus passed downwards freely after the first twelve hours, and there was no pain and no abdominal distension. The urine was abundant and free from albumen. The bowels were moved on the fifth day, after one drachm of sulphate of magnesia had been given. The lower bowel then became somewhat irritable. The patient once or sometimes twice daily would have a strong desire to defæcate, with forcing pain, but no power of expulsion. In this condition a small enema produced an evacuation of the bowels, and the patient was for the time quite comfortable. The lower end of the wound was dressed daily after the fourth day, and the separation of the slough seemed to go on in a perfectly satisfactory manner. On the eighth day the temperature rose from  $100.6^{\circ}$  to  $103.2^{\circ}$ , and the

pulse to 128. There was at this time nothing unhealthy to be seen about the wound; the patient was absolutely without pain, and said she felt very well. The urine had become alkaline and offensive, and this was the only condition I could find that seemed to account for the rise of temperature. After the application of ice to the head the temperature came down, and next morning it was  $100.2^{\circ}$ , the pulse being 96. Dilute nitric acid was given, and the urine quickly became acid again. The temperature gradually descended, and on the eleventh evening the highest was  $100.8^{\circ}$ , while next morning it was  $99^{\circ}$ . The pulse came down to 84, but was still very excitable. I had now no fault to find with the condition of the patient, except that she was very anæmic, which seemed to account for the pulse condition, and that the lower bowel was still irritable. The wound looked healthy, the wire round the pedicle was quite loose, and was removed without any difficulty on the eleventh morning. On the morning of the twelfth day the patient said she was very comfortable. The skin, which had been rather dry since the operation, and which the patient said seldom acted much, was decidedly moist. I noted this and thought it curious, but, with the other conditions as satisfactory as they seemed, it caused me no alarm. In the early afternoon the patient's respirations became very hurried,—over 50 to the minute; the pulse rose to over 120, and she complained first of pins and needles, and later of extreme pain in her left leg, which quickly became swollen. The unfavorable symptoms rapidly increased in severity, the patient passed into a state of collapse, and died at 8 p.m. on the twelfth day after the operation. The temperature at the end did not rise above  $99.6^{\circ}$ .

At the autopsy there were found slight recent adhesions in the immediate neighbourhood of the operation wound, but there was no lymph and no fluid in the peritoneal cavity. The pin supporting the stump of the uterus outside the abdomen had been removed after death,

and the pedicle had dropped down into the pelvis, the left iliac vessels being exposed. The vein was distended with clot, and contained bubbles of gas. The right side of the heart was distended with soft plum-coloured post-mortem clot containing a few bubbles of gas. The lungs were "remarkably bloodless, but otherwise normal."

This death was more directly due to the hysterectomy than to the ovariectomy, but as it was the ovarian tumours that necessitated the operation the case comes under the title of my paper. Phlegmasia dolens after an ovariectomy is not a very rare complication, but this is the only fatal case that has occurred in my own practice, and I do not remember having seen any fatal case in Mr. Knowsley Thornton's hospital work during the seven years that I assisted him in it. When the abdomen is completely closed the iliac veins may become plugged without any warning whatever, and without any indication that septic mischief exists. In the case I have just related, it, however, seems evident that septic contamination spread directly from the sloughing uterine stump to the iliac vein.

Before this case occurred I had been in the habit of clipping away the sloughing pedicle as quickly as possible, but now I always let the parts separate with very little interference and few dressings. The exact adjustment of the peritoneum of the anterior abdominal wall round the stump of the pedicle behind the serre-nœud wire is the most important factor in preventing such fatalities, but when the broad ligament has been extensively opened up there is necessarily great difficulty in securing the parts satisfactorily.

The next patient was sent to me by Mr. Manley Sims. She was a single woman, sixty years of age, in whom menstruation had always been free, but had never given any trouble. It ceased at the age of forty-six, but at the age of about fifty-four and since then she had had a discharge, sometimes red, sometimes pale, and sometimes of a dirty-brown colour and offensive. Otherwise the

patient was a healthy active woman till the end of June, 1893, when an enlargement of the abdomen was first noticed. It increased rapidly, and was accompanied by pain, constipation, and emaciation. When I first saw the patient on August 28th, 1893, the abdomen was greatly distended, the skin over it was tense and glistening, and the superficial veins were enlarged. The parts were soft above, but resistant around and below the navel. The note on percussion was resonant in the loins and upper abdomen, but dull anteriorly, and the dull area moved very slightly with changes in the position of the patient. Through the vagina a hard nodular tumour was felt, filling the pelvis, firmly fixed, and pushing the parts very low down. The cervix admitted about half an inch of my forefinger; it felt soft and friable, and immediately above and behind it there was a very hard protuberance, about the size of a walnut, standing out from the main growth. I did not attempt to pass a sound, as it seemed quite as likely that I might push it through diseased tissues as into the cervical and uterine canals. I believed that the patient had advanced malignant disease of the uterus, a growth, probably also uterine, in the lower abdomen, and a large quantity of fluid free in the peritoneum. Mr. Sims, who had seen the patient twice before, felt sure that there was an ovarian tumour in addition to the uterine condition. An exploratory operation was therefore performed in a nursing home on August 31st, 1893, when I found and removed a very friable, multilocular, ovarian tumour, which had numerous small cysts with much solid matter between them. The solid part weighed three pounds. It had a rather wide attachment to the right broad ligament, and was floating in a large quantity of thick glutinous fluid that had exuded from its surface. Twelve pints of this were measured, but much was lost. The uterus nearly filled the pelvis, and rose about two inches above the level of its brim. It had grown into and expanded the left broad ligament, and its outline was smooth, rounded, and

soft, with numerous hard nodular places felt in its walls, but these did not project. The left tube and ovary were healthy, but bound down by old adhesions. As much as possible of the ovarian fluid was removed, and the abdomen was closed without drainage. For the first four days after the operation the highest temperature and pulse were respectively  $101.2^{\circ}$  in the vagina, and 106 to the minute. On the evening of the third day there were some abdominal pain and distension, both of which were removed by an action of the bowels following an enema on the fourth morning. The skin and kidneys acted freely, the vaginal discharge was slight and not offensive, and the patient slept well at intervals. On the afternoon of the fourth day the vaginal discharge ceased; the temperature, which had been  $100.2^{\circ}$  in the morning, rapidly rose, and the patient had a severe rigor commencing at 1 a.m. on the fifth day. An hour and a half later the temperature was  $104.8^{\circ}$ , and the pulse was 140. Soon after the rigor ceased I dressed the wound, which was quite free from any sign of irritation, the abdomen being soft and flat. I attempted to pass sounds and catheters of various shapes into the uterus, but failed to do so. Shortly after this, however, the uterine discharge returned, and the temperature fell to  $100.6^{\circ}$ , and the pulse to 112, nine hours after the beginning of the rigor. The remainder of the fifth and the first half of the sixth days were passed without trouble, the patient's condition being greatly improved, but in the afternoon of the sixth day the uterine discharge again ceased, and the patient had another attack similar to that just described, except that there was no rigor. All my efforts to pass an instrument into the uterus failed, and on this occasion the discharge did not return. Flatus ceased to pass downwards, the abdomen became distended, the patient became delirious, and she died at 8 p.m. on the seventh day after the operation, the temperature being then  $108^{\circ}$ .

At the post-mortem examination there were signs of



very slight general adhesive peritonitis, with intestinal distension, but there was no exudation of fluid or of visible lymph from the peritoneum. The uterine cavity was dilated, and contained ten or twelve ounces of mucus, its lining membrane being much thickened. In its walls there were two old hard fibroid tumours as large as Tangerine oranges, and many others of smaller size. None of these caused any prominence on the peritoneal surface of the uterus, but most of them bulged into its cavity, which thus had a very irregular outline. One fibroid was outside the uterine substance low down in the left broad ligament, whilst another was in the posterior wall of the cervix, and had evidently been the chief cause of the dilatation of the womb, and of the difficulty in passing an instrument through the cervical canal. If I had not been under the impression that the uterine disease in this case was malignant, I should probably have made a more determined effort to find, or make a way into the uterus after the rigor, and free drainage might have saved the patient's life, but I did not know that there was such a considerable cavity in the womb, and to apply any great force to a malignant uterine growth would have been to jeopardise any chance of recovery that existed. The conditions found were new to me.

The last of these patients was sent to me by Mr. Knowsley Thoruton and Dr. Boyce of Maidstone. She was thirty-nine years of age, and she had first noticed a lump "like a ball" in her abdomen in 1889. This gradually enlarged, and when I saw her in 1891 it had been growing more quickly for a few months. There was a large fluctuating oval tumour in the right side rising out of the pelvis well above the level of the umbilicus, and reaching a little beyond the outer edge of the rectus muscle on the left. Attached to the lower portion of this tumour, in the left groin, below the level of the anterior superior iliac spines, was a hard nodular swelling, the whole being firmly fixed. The cervix was very high and far forward in the pelvis,

the os being almost out of reach. The fundus was not definable. Behind the cervix a hard rounded mass distended the pelvis far below the os uteri, down nearly to the anus. I diagnosed an ovarian tumour opening up the broad ligaments, or possibly a cystic fibroma of the uterus.

On November 24th, 1891, I opened the abdomen, and found that the bulk of the tumour had the colour of a healthy uterus, and appeared to be an enlargement of that organ. I did not feel certain whether it was a very soft fibroid or a cystic growth. The mass in the left groin also appeared to be uterine. It was within the broad ligament, was covered by very large veins, and was of stony hardness. There were several small nodular growths under the peritoneum of the anterior abdominal wall, and the glands along the brim of the pelvis on the left side were distinctly enlarged. I came to the conclusion, and Mr. Doran, who was assisting me, agreed that the tumour was a malignant uterine growth and irremovable. I therefore immediately closed the incision without investigating the position of the ovaries or the posterior connections of the tumour, as this would have necessitated a considerable enlargement of the wound.

There was no trouble during convalescence, and the patient went home on the eighteenth day. The general condition and the state of the bowels were much improved for a time, and when I saw the patient four months later the abdominal measurements were smaller than they had been before the operation, the pain also being relieved. This diminution in size and absence of pain were, I think, due to an improved condition of the bowels. The patient continued fairly well for about eighteen months, but her abdomen gradually enlarged and she became weak and thin. About June, 1893, she increased rapidly in size, and again suffered from attacks of severe pain. About this time, as she informed me, she was told by a medical man that there could be no difficulty about removing her tumour, and no doubt that she would get well if this were done. She therefore asked Dr. Boyce

and me to reconsider her case, and she was again admitted to the Samaritan Free Hospital in October, 1893. The tumour had then the same shape as before, but it was much larger. The main part fluctuated distinctly. It reached above the costal margin, and well over to the left side, its upper end being almost central in position, whilst the smaller portion on the left side had risen nearly up to the level of the umbilicus. The superficial glands in the left groin were slightly hypertrophied. In the pelvis the growth was larger and even lower down than before; the cervix uteri was quite out of reach of the finger. I could not feel any of the subperitoneal nodules that were found at the first operation except one, which was close to the old scar. The superficial veins in the left groin were slightly enlarged. The general condition of the patient was, however, altogether inconsistent with the view that the tumour was a malignant one. I therefore determined, after consulting Mr. Thornton and other colleagues, and with their approval, to take away the tumour if possible. The patient was extremely anxious to have this done, although the very serious nature of the operation was put before her in the clearest language.

On October 25th, 1893, I reopened the abdomen, a cystic growth the size of a bean in the scar of the first wound being cut into and excised. Some slight adhesions to the anterior abdominal wall caused by the first operation were easily broken down with the finger. There were no other adhesions of the peritoneum, but this membrane was reflected from the tumour to the abdominal wall about two and a half inches above the pubes in front, well above the promontory of the sacrum behind, and correspondingly high at the sides, so that the whole of the pelvic peritoneum and part of the peritoneum of the lower abdomen had been separated from its proper connections by the new growth below it. There was no line of demarcation either as regards colour or form between the right tumour and the front of the uterus, of which it still seemed to be an enlargement. The

growth on the left side had now, however, all the appearances of a thin-walled ovarian cyst, completely covered over by the broad ligament. Its anterior surface was continuous with the larger muscle-coloured growth, the limits of the two in front being marked by the colour alone, and a shallow sulcus separating them above. I tapped the larger growth, which had walls quite half an inch thick, and contained opaque yellowish-grey fluid. When reduced in size the growth was brought outside the abdomen, but the thick cyst-wall would not collapse sufficiently to allow of this till I made the ventral incision about eight inches long. The full extent of the base from which the tumour had to be enucleated was now seen. After dividing the peritoneum the growth was shelled out of the right broad ligament, but this was not easily done, as, although it did not go deeply into the pelvis, the tumour was attached so firmly to the neighbouring structures by strong bands of connective tissue that a knife or scissors had to be frequently used. The smaller cyst on the left side extended very deeply into the pelvis, squeezing all the organs into the smallest possible space. I attempted to get this cyst out unopened, because it is easier to separate a full cyst than a collapsed one from neighbouring structures, but, when I had nearly succeeded, it burst, several pints of dark-coloured fluid escaping. I had now to separate the rectum from a very thin and of course quite flaccid cyst-wall at the bottom of the pelvis, the two being firmly attached by the irritation of prolonged pressure, and my action being hampered by the presence of the uterus and collapsed tumours. Although I was keenly alive to the danger, an opening was made in the rectum. I got both the tumours completely out, passed the wire of a serre-neud round the stump formed by the neck of the uterus, and cut away the parts beyond it. During the enucleation the left ureter had been exposed for about six inches, and the right for about five inches. I sewed up the tear in the rectum carefully, fixed the stump of the uterus outside the peritoneum in the lower

angle of the wound, and closed the incision, placing a rather short drainage-tube in Douglas's pouch. In spite of every effort to keep the patient warm and to sustain the heart's action she barely survived the operation.

The mass removed consisted of two ovarian cysts, containing papillomatous growth, which had developed in the broad ligaments and raised the uterus high up into the abdomen in front of and between them. Above and behind, the peritoneum was reflected from one cyst on to the other, and was completely removed from the back of the uterus.

At the autopsy the kidneys, especially the left, were dilated, but otherwise the organs were healthy. In this case the patient did not lose a great deal of blood,—not nearly so much, for instance, as was lost in the second case above related. Hence the hæmorrhage alone would not account for the death, which I attribute to shock from the extensive laceration of important structures, especially the nervous plexuses in front of the sacrum.

Such deaths as this are to be avoided by the application of artificial warmth, by wasting no time in operating, and I think in some cases by transfusion; but it does not seem yet to be settled whether it is advisable to transfuse in cases of shock. I devoted my whole attention in this case to trying to keep the patient warm and to prevent the heart from stopping. I fear that, in abdominal surgery, deaths such as this one must occasionally happen from the mere magnitude of the operation unless we are prepared to take the responsibility of refusing surgical treatment in all very dangerous cases—a course particularly to be condemned as regards cases of ovarian tumour, because recovery from the worst cases may be complete. In the case under consideration, for instance, there was nothing in the patient's history, or in the conditions found, that would have prevented her from enjoying long life if the immediate risks of the operation had been overcome; whereas if I had refused operation

death was certain, and tapping would probably have resulted in a general infection of the peritoneum by papillomatous growth.

It is, of course, impossible to say whether the result would have been favorable if I had gone on and removed the tumour at the first operation. Hæmorrhage would probably have been greater then, but the enucleation would have been rather less deep, and the condition of the kidneys was no doubt better. I think, however, it is quite possible that my mistake gave the patient two more years of life, eighteen months of which were spent in comparative comfort. Just before the exploratory operation the patient had suffered much pain in the left groin accompanied by febrile symptoms, an attack which was not specially brought under my notice till afterwards. I think the inflammation causing these symptoms had induced a fresh effusion of fluid into the left ovarian cyst, and this accounted for the great vascularity and stony hardness of this part of the tumour at that time—the conditions which chiefly led to the error in diagnosis.

I have now related all that is essential in the histories of the above cases, so as, if possible, to convey an accurate impression of the progress of each patient towards a fatal issue, and I have not attempted to minimise the importance of any accident or mistake that had occurred. I may, perhaps, be permitted to add that the conditions as observed before the operations and from day to day during the after-treatment of the cases were, in some instances, by no means so clear as they are now.

I attribute two of the deaths (Nos. 3 and 6) to shock; two (Nos. 1 and 2) primarily to obstruction of the bowels; and two (Nos. 4 and 5) to septicæmia brought about by unavoidable complications. Intestinal difficulties and septicæmia are the most important dangers of abdominal surgery. As regards the first, increasing experience confirms me in the views I have already expressed. As to septicæmia, it is a matter of special satisfaction to me

that I have not to record any case in which septic infection, followed by death from acute peritonitis within three days, has been communicated to the patient at the time of operation, as sometimes happens when no antiseptics are used, or when they are used without sufficient care. Such rapid deaths from septicæmia occur in simple cases as readily as in difficult ones, but they may be prevented with almost absolute certainty by the intelligent use of antiseptics and by a never-failing watchfulness on the part of the surgeon and his assistants as regards the patient's surroundings. Watchfulness and care should also prevent those fatalities which Mr. Mayo Robson<sup>1</sup> has described as "preventable,"—namely, those due to slipping of ligatures, and those due to putting patients into insanitary wards. The sanitary condition of a ward as regards the drainage of the hospital should, of course, be as perfect as it can be made. Apart from this, one of the chief safeguards against septic infection is the placing of all patients, whose peritoneums have been opened, in rooms set aside for the purpose, during convalescence. Success may often be obtained without this precaution, but no amount of statistics can overcome the fact that the peritoneum is extremely prone to contract septic inflammation. Hence, as a matter of prudence, no surgeon should run any risk, if he can possibly avoid it, of allowing his abdominal section cases to be brought into an atmosphere contaminated by the presence of a number of sick people. I repeat that under such circumstances deaths from septicæmia are as likely to occur in simple as in complex cases, and that septic peritonitis after a simple ovariectomy is a preventable disease.

Septicæmia coming on later, and due to some cause of the nature of an unavoidable complication, is more difficult to prevent; but the more clear the records of the way in which death follows operations such as I have recorded, the more likely is it that surgeons will prevent similar results in the future. I therefore venture to hope that the

<sup>1</sup> 'Brit. Med. Journ.,' 1894, vol. ii, p. 977.

histories I have related may help others in dealing with difficult cases, and also that valuable assistance may be obtained from the discussion which I trust will follow.

I do not wish to divert remark from any points in the cases related which may interest the Fellows, but I think it may be convenient if I suggest for discussion the following propositions which seem to be in accord with our present knowledge of the subject under consideration, and to be supported by the records I have given.

1. That in dealing with ovarian tumours it may be impossible to make an exact diagnosis, and it may be impossible to remove successfully even a non-malignant growth. Hence the selection of cases for operation, which many men profess not to make, is the most difficult and perhaps the most important duty of the surgeon in this branch of his art.

2. That septicæmia is the most common danger of abdominal surgery, but that septicæmia, in uncomplicated cases of ovariectomy, is preventable by the use of anti-septics and by care; whereas in certain complicated cases its avoidance is difficult, and may be impossible.

3. That functional and mechanical disturbances of the bowels are the special dangers of abdominal surgery.

I would further submit for consideration that if there seems to be any possibility at all of removing an ovarian tumour with success, it is the surgeon's duty to explain clearly the circumstances of the case to the patient, and *if she then wishes it*, he should be ready to accept the responsibility of operating, even if he has but little hope as to the result, rather than leave the woman to the natural issue of her disease. The latter course is so hopeless in cases of ovarian tumour, and the matter is so much more important to the patient than to the surgeon, that if there be any uncertainty as to how to act, the patient and her friends ought to make the final decision.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. vii, p. 44.)



# VARIETIES OF INTESTINAL OBSTRUCTION DEPENDENT ON GALL-STONES,

WITH A SERIES OF CASES

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By intestinal obstruction from gall-stones is usually understood the impaction of a large concretion in some part of the small intestine producing a mechanical block; but this is only one of the several varieties of obstruction of the intestines dependent on cholelithiasis. There are clearly four classes of obstruction depending essentially on gall-stones.

1st. Acute obstruction caused by local peritonitis in the region of the gall-bladder leading to paralysis of the bowel. Of this variety I have seen several examples, all of which recovered without operation.

2nd. Volvulus of the small intestine, dependent either on the violence of the colic caused by the attack of gall-stones, or on the contortions induced by the passage

of a large concretion through the small intestine. I append the notes of two cases of this kind, in both of which operation was performed, recovery following in each after simply untwisting the volvulus.

3rd. Mechanical obstruction due to the passage of a large concretion through the small intestine, of which I shall describe one case, and by permission mention another, both of which recovered after enterotomy.

4th. Obstruction coming on after the original cause has disappeared, and depending on adhesions left by local peritonitis due to gall-stone attacks, or on narrowing caused by the healing of a fistulous opening through which a large gall-stone has made its way into the intestinal tract.

Of this variety I do not propose to give detailed examples, as they are only remotely due to the cause in question, and may be more usefully discussed in a paper on intestinal obstruction from general causes.

CASE 1.—Early last year I received a telegram from Dr. H. asking me if I could receive a patient, Mrs. —, æt. 60, at a surgical home for immediate operation for acute intestinal obstruction, fæcal vomiting having been present for three days, and medical treatment having failed to relieve.

On arrival the patient was too exhausted and ill to bear operation, and morphia was administered to relieve her distress and combat the collapse due to the intense pain. Rectal feeding was at once begun in order to maintain the strength, and Ext. Bellad. was given every four hours in  $\frac{1}{4}$ -grain doses. The patient forthwith began to improve, and a clear history of cholelithiasis was obtained, the present attack, the patient said, differing in no respect, except in severity, from those she had frequently had on former occasions. There was marked tenderness over the gall-bladder, particularly at a point one third of the distance in a straight line between the ninth costal cartilage and the umbilicus, together with some

swelling in the right hypochondrium, slight tympanitic distension of the abdomen generally, some jaundice, and the history of a sudden onset followed by two or three slight ague-like attacks. Flatus passed the same night, and continued to pass at intervals.

After two days the bowels were freely relieved after a large enema had been administered. No large gall-stone was discovered, but several small concretions which had evidently passed through the common duct. She returned home in the third week, and has remained well.

CASE 2.—Mrs. R—, æt. 56, was admitted on July 18th, 1893, into the Leeds Infirmary with symptoms of acute intestinal obstruction of three days, and fæcal vomiting of twenty-four hours' duration. The patient was jaundiced and was in very great pain, the pain having begun over the gall-bladder, radiating thence over the abdomen, and through to the right scapular region. She gave the history of having had numerous gall-stone attacks during the previous fifteen years, but she had never been so severely affected as on the present occasion.

A morphia injection, followed by  $\frac{1}{4}$ -grain doses of Ext. Bellad. every four hours, and rectal feeding, soon gave relief to the urgent symptoms, and the bowels were moved on the third day, after which recovery was uninterrupted. On October 21st, 1893, the patient having completely recovered from the obstruction, but the spasmodic pain followed by jaundice having recurred, I opened the abdomen, and found numerous adhesions of the colon and duodenum to the gall-bladder and bile-ducts.

Cholecystotomy was performed, and six stones were removed, several others in the common duct being crushed between the finger and thumb.

The patient was discharged cured in a month, and has been well since.

These two will suffice as examples of the first class of obstruction.

CASE 3.—As this case is reported on p. 92 of my work on gall-stones, I will only mention it, and not describe it in detail.

Acute intestinal obstruction in a woman of sixty-eight operated on November 12th, 1890, by laparotomy, on the eighth day of the obstruction, a volvulus of the small intestine being discovered and untwisted. Bowels moved by enema on the sixteenth day after onset of obstruction and eighth day after operation, and a large gall-stone 3 inches in circumference and  $1\frac{1}{2}$  inches long was passed, this being manifestly the cause of the obstruction, and secondarily of the volvulus.

The patient returned home on the twenty-sixth day, and remained quite well when heard of a year subsequently.

CASE 4.—On March 13th, 1894, I received a telegram from Dr. Lownds, of Doncaster, asking me to go prepared to operate on a case of acute intestinal obstruction. I found a Mrs. O—, aged 62, suffering from acute obstruction of six, and fæcal vomiting of two days' duration, the onset having started like a gall-stone attack with pain over the gall-bladder in the beginning, but subsequently in the umbilical region.

She gave a history of having suffered from attacks of gall-stones for several years, some of which had been followed by jaundice, and from the mode of onset of the present seizure, and the slight jaundice following it, she was quite sure the attack was one of her old seizures at the commencement.

From the persistence of the fæcal vomiting, the presence of visible intestinal peristalsis, and the pinched and anxious countenance, with the absence of relief by ordinary medical means, operation was decided on.

Laparotomy was performed, and volvulus of the small intestine being found, the loop of gut, which was much congested, was untwisted and the abdomen closed. Flatus passed the same day, and the bowels were opened

the next. The wound healed by first intention, and recovery was uninterrupted.

These two cases are illustrative of the second class of obstruction.

CASE 5.—On September 13th, 1894, I received a telegram from Dr. Tempest Anderson and Dr. Raimés, of York, to go prepared to operate on a case of acute intestinal obstruction, but on arrival word was brought to the station that the patient was in a state of collapse, and might be dead on our arrival. Fortunately, however, we went, and as a result of a morphia injection administered by Dr. Raimés before our arrival the pulse had recovered itself, and she was a little better. The patient, a lady of fifty, gave a characteristic history of gall-stone attacks without jaundice for over ten years, but during the past year she had been much better until Saturday, September 8th, when she was seized with violent pain in the centre of the abdomen of a colicky nature, which was slightly relieved by opium; the pain, however, soon recurred, and was accompanied by vomiting, which became fæcal on Monday, the 10th. Despite morphia and other means the symptoms persisted, and on Wednesday, the 12th, chloroform was administered and abdominal massage with abdominal succussion was employed, but without material relief.

When I saw her at 10.30 on the evening of Thursday, the 13th, her pulse was rapid and intermittent, and she looked extremely ill, though she was temporarily relieved by the morphia which had been given a little time before our arrival. There was no distension of the large bowel, but visible coils of small intestine pointed to some obstruction in the lesser gut, and we all agreed that operation was our only course. At 1 a.m. on the 14th September the abdomen was opened by a 1½-inch incision below the umbilicus, and almost immediately a hard lump was felt inside a coil of small intestine at the bottom of Douglas's pouch. This loop was brought through the abdominal

incision and surrounded by gauze wrung out of carbolic lotion.

After emptying the gut by pressure, Dr. Anderson grasped the proximal and distal ends between his fingers and thumbs; the bowel was then incised and the stone extruded, the opening in the gut being closed by a continuous catgut suture for mucous membrane, and a continuous silk suture for the serous coat. The surface of the bowel which had been exposed was then bathed with boracic lotion and returned, and the abdominal incision closed in the usual way.

From beginning to end the operation occupied but twenty minutes, and the patient was put into bed in much better condition than she was in before the operation. The wound healed by first intention, and there was nothing to chronicle in the after progress, the patient being now quite well. The stone weighed  $1\frac{1}{4}$  ounces when dry, and measured three inches in circumference in one direction, and  $4\frac{1}{4}$  inches in circumference lengthwise.

CASE 6.—By the kind permission of my friend Dr. Everley Taylor I am permitted to mention a recent case of acute intestinal obstruction, in which he removed successfully by enterotomy a concretion from the ileum which it was completely blocking. I believe the gall-stone, of which I show the photograph, is the largest which has been successfully removed, if it be not the largest on record as having been found in the intestines.

These cases illustrate the third class of obstruction.

When I began to write this paper I thought intestinal obstruction from gall-stones to be not very uncommon, but in order to ascertain whether this was so or not, I addressed a letter to the registrars, or to members of the staff of a number of the largest metropolitan and provincial hospitals, asking how many cases of intestinal obstruction dependent on gall-stones had been admitted during the past year. The answers I received, and for which I sincerely thank my correspondents, represent about

80,000 in-patients treated during the year ; and out of this large number, my own cases excepted, only four instances of gall-stone obstruction were reported : two of these were at the London Hospital, one at Guy's, and one in the Manchester Royal Infirmary, which shows that this form of intestinal obstruction is far from frequent, and certainly less common than I had expected.

Having related the cases, which may perhaps be found to present points of interest, I propose to make a few remarks on the diagnosis of the several conditions, and then to discuss the question of treatment.

In the first class (cholelithic attack) the diagnosis will not as a rule be difficult, as the history of the occurrence of previous attacks of spasms, at times—though not of necessity—followed by jaundice ; the similarity to these of the commencement of the attack in question ; the severe and persistent pain, at first localised to the right side of the abdomen ; the absence of distension at the commencement, and then the occurrence of distension on the right side only, becoming general later ; the lateness of the onset of fæcal vomiting, and then only after continued retching ; the existence of collapse at an early stage owing to the severity of the pain, which is usually relieved by a morphia injection ; the usual absence of visible peristalsis ; and lastly, the onset of jaundice if the concretion have reached the common duct, afford so much guidance that error will not often occur, especially if the patient be a woman of middle or old age. But that difficulties may arise is shown by the cases which I have mentioned.

In the second class of cases (volvulus) a positive diagnosis is probably, for the most part, out of the question except after the abdomen is opened, as volvulus of the small intestine is an extremely rare event, and we know that, as in the third case, a large gall-stone may quietly ulcerate its way into the gut without any preliminary warning, the symptoms only arising when the concretion is passing through the small bowel ; but in

both cases related, in addition to the signs of acute obstruction, there was a well-marked localised swelling near the umbilicus, becoming hard at intervals when the paroxysms came on, pointing to the site of the obstruction; and in the fourth case there was not only the previous history of cholelithiasis, but the characteristic onset of a gall-stone attack followed by acute symptoms.

The third class of cases (impaction in small intestine) has given rise to much discussion on previous occasions, and I would only here point out that although in the fifth case related I was able to make a probable diagnosis from the history of previous gall-stone attacks extending over several years, yet in many cases there is absolutely no previous history to guide one, and it is quite impossible to say whether or not the attack is one dependent on the cause in question, or on a volvulus or band or internal hernia which, if left, must almost inevitably lead to death, and that speedily. The age and sex, together with the history of chronic dyspepsia and pain in the hepatic region, are, however, well worth bearing in mind, as well as the early and persistent vomiting and visible peristalsis limited to the small intestines.

In the fourth class the symptoms are usually subacute or chronic, and have often been preceded by long-continued constipation or partial obstruction; coils of intestine will usually be visible; and where the site is in the hepatic flexure of the colon, as in a case I have lately seen, the vermicular action starting in the small intestine, and extending to the cæcum and ascending colon, stopping in the region of the gall-bladder, pointed to the stricture which was the cause of the trouble.

*Treatment.*—The first class of cases will, as a rule, yield to medical treatment, and it can seldom be necessary or even advisable to resort to operation during the seizure, though, as in the second case, a subsequent operation may be required.

In the second class (volvulus), it would seem to me that operation holds out the only hope of success, as the



obstruction being mechanical, nothing short of remedying the cause can be expected to be of use.

The third class of cases (impaction in small gut) has given rise to much discussion, as it is an undoubted fact that large concretions have safely passed through the alimentary canal after obstruction of several days' duration, but when it is borne in mind that there are no symptoms peculiar to this form of obstruction, and that the course pursued by an obstruction by a band or by an internal hernia may be exactly the same as in gall-stone obstruction, the surgeon who waits beyond the period when an operation may be undertaken with every hope of success, is incurring a very serious responsibility.

In my own case related above, medical means as well as manipulation under an anæsthetic had been fully tried, and her condition after five days' obstruction with three days of fæcal vomiting was such that further delay would, in my mind, as well as in the opinion of the able physician and surgeon with whom I was associated in the case, have been very unwise.

With regard to the method of treatment after the abdomen is opened and the cause found : if the gall-stone can be crushed or needled through the intestinal coats, such may be done, but I should prefer to incise the gut and perform enterorrhaphy, as it can be done very quickly and with very little damage to the bowel.

Should the patient be too ill to bear a search being made for the obstruction, enterotomy or short circuiting might be performed in order to give temporary relief, the cause of the obstruction being afterwards removed, if it were not effected naturally. As to when operation should be performed, that is part of a general question which each surgeon will have to answer for himself in every individual case, as no definite rule can possibly be formulated which will apply to all cases. The surgeon will, as a rule, not be called in before decided symptoms of intestinal obstruction have manifested themselves, and until medical means have been fully tried ; in such cases it would seem to me

to be idle waste of time to delay surgical intervention until the patient is so exhausted that operation is only undertaken as a "dernier ressort" when the patient is almost moribund. If, however, the case be seen at an earlier stage, morphia will have to be given to relieve the pain, and I have usually recommended Ext. Belladonnæ in  $\frac{1}{4}$ -grain doses every four hours, the stopping of all food by the mouth, and the administration of one large siphon enema or more given slowly with the buttocks elevated. If relief does not speedily follow, and the diagnosis is not clear, chloroform anæsthesia may assist in two ways: in the first place it enables a thorough examination of the abdomen, and at times a diagnosis of the cause to be made; and secondly, the manipulation, if made methodically, may reduce a hernia or volvulus, or may possibly help onwards an obstruction.

This failing, and the symptoms persisting, I should operate, and at this comparatively early stage I feel sure with every prospect of success.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. vii, p. 52.)

# THE CAUSATION OF ÆGOPHONY.

BY

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THE subject of ægophony as a physical sign is one of some interest, though it has perhaps declined in importance since it has been discovered that a small needle may be inserted into the chest, and a certain diagnosis of liquid effusion can be made, with but little pain and no risk to the patient ; and there may be some who think with the late Dr. Anstie<sup>1</sup> that " it is, in truth, one of the *fancy* signs of pleurisy, interesting rather than useful." But it is at least a natural phenomenon, the explanation of which has been much debated ; and as a physical sign it is very desirable to know whether it has any pathognomonic significance or not.

If the morbid sound is due to the presence of liquid, and can only be explained by theories involving the presence of that medium, then the sign must be absolutely diagnostic of effusion. If, on the other hand, the sign has ever been unequivocally heard when liquid has been absent, then we must look for an explanation to

<sup>1</sup> Russell Reynolds' 'System of Medicine,' vol. iii, p. 929, art. "Pleurisy."

physical conditions independent of liquid. There can of course be no doubt that liquid is nearly always present in the cases in which ægophony is heard; but it must not be forgotten that liquid cannot exist in the pleural cavity without considerably modifying, by its pressure, the condition of the corresponding lung; and therefore the condition of the lung must be taken into account as well as the properties of the fluid in any inquiry into the nature of the phenomenon.

I need not go far into the history of ægophony. Laennec, who first described it, attributed it to the compression and flattening of the bronchial tubes, aided by a thin layer of fluid, which could be set in movement by the vibrations of the voice. Skoda would not allow that the flattening of the bronchial tubes could produce a tremulous sound, which, according to him, must be due to the impulses of a solid body upon another body, whether solid, liquid, or gaseous.<sup>1</sup> And he accordingly regards it as "probable that in most cases the wall of the bronchus, within which the air resonates, recoils in impulses upon the contained air, and so gives rise to the tremulous sound." But he also thinks it possible that sometimes a particle of mucus, which incompletely covers the opening into a bronchus, may act like the reed of a musical instrument, and cause the sound in question.<sup>2</sup> Nor does he allow that the sign is distinctively characteristic of liquid effusion in the pleura.

Since this work of Skoda's, the researches of Helmholtz have so increased our knowledge of acoustics, that we are in a better position than the older physicians for forming an opinion as to the nature of this curious sound; and the late Dr. Stone, especially, did good service in applying the principles of harmonics laid down by Helmholtz to the solution of the difficulty.

The prevailing view amongst English writers at present is that liquid in the pleura is mainly responsible for the

<sup>1</sup> 'Abhandlung über Perkussion und Auskultation,' 6th edit., 1864, p. 77.

<sup>2</sup> Loc. cit., p. 70.

production of ægophony ; and that it produces it either by intercepting the fundamental note and lower harmonics through its inferior conducting power (Douglas Powell), or by its multiplying the media between the lung and the surface (Stone, Bristowe, Fagge), or by its supplying additional secondary vibrations to those passing through it (Douglas Powell).

I have already been led to express an opinion on the subject in my manual of 'The Practice of Medicine,' of which the first edition, published in 1890, contains the following passage (p. 348) :

"It has been shown by Dr. W. H. Stone to be due to the suppression of the fundamental note and the lower harmonics of the vowel sounds, while the higher harmonics are transmitted in an accentuated form. The most common cause is undoubtedly the presence of liquid in the pleura ; and this suppression of the lower tones is attributed to their inability to pass through—that is, to set up vibrations in—the layer of liquid. But it appears to me it is better explained by supposing that the bronchial tubes are modified by compression, so that whereas they normally resonate the lower harmonics, now they can resonate only the higher harmonics ; thus the former are stopped, not at the fluid, but at the bronchial tubes."

In later editions the passage remains substantially the same.

The object of the present communication is to state in support of this belief such facts and reasons as I could not introduce into the work referred to, and further to supply an omission which is common to my own description with that of Dr. Stone, and that is, to adequately account for the tremulous or bleating quality of the ægophonic sound.

I shall endeavour, then, to show that—

1. Ægophony is no more than, nor different from, ordinary musical *discord* or *dissonance*.
2. This discord is the result of *beats* occurring between the higher harmonics of the note uttered by the patient.

3. The beats constituting the discord are audible because the higher harmonics are reinforced, while the fundamental tone and lower harmonics are suppressed.

4. Both the reinforcement and the suppression are due to modifications of the bronchial tubes, which cause them to resonate the higher harmonics and not the lower.

The two characteristic features of ægophony are the tremulous or quavering quality of the sound, and its high pitch. It is essential to understand that the first of these qualities is musical discord, and that it has no special relation to liquid as such, nor to the mere fact that vibrations take place in this or that medium or structure, because all sounds of whatsoever character, musical or noisy, are the result of vibrations. It would hardly be necessary to insist on this were it not that some authors have written in such a way as to make one suppose that they thought a quavering sound resulted solely from a simple vibrating motion of solid or liquid.

For instance, Guttman<sup>1</sup> says, "Ægophony is most probably produced by vibration of the flattened, compressed bronchi. This vibration is excited by the voice, and transmitted to the thin layer of fluid which, at the upper part of the exudation, lies between the lung and the chest wall. *This tremulous movement of the sides of the bronchi gives the voice sound a quavering interrupted character.*"

But the theory of musical discord and beats is really wanted to explain the tremulous, quavering, nasal quality of ægophony. It will not be becoming for me to do more than re-state the propositions which may be found in any text-book of acoustics as follows :

That beats are produced when two notes are sounded together which differ by a small number of vibrations from one another.

That beats are sounds resulting from the repeated coincidences of the waves of sound, which coincidences

<sup>1</sup> 'Handbook of Physical Diagnosis,' New Syd. Soc., 1879, p. 175.

will be repeated as many times in a second as the difference between the vibration-numbers per second of the two notes.

That when the beats reach thirty-three per second the effect upon the sound is disagreeable to the ear, and becomes more so as the number per second increases.

That the maximum of disagreeable effect is reached when the number is about 70.

That the effect is again lost when the beats number more than 133 per second.

That the number of beats required to produce this discord varies in different parts of the scale, *i. e.* according as the vibration-numbers of the notes themselves are greater or smaller.

That beats are produced not only between the vibrations of the fundamental tones, but between the fundamental tones of one note and the harmonics of the others, and between the harmonics of the two tones.

That a compound tone may be harsh or dissonant in itself, if it contains very high or loud harmonics.

Now it is the fact of the identity of the ægophonic sound with a musical discord, and the necessity of explaining the former in this way, which, with one exception, writers on the subject hitherto have entirely failed to emphasise and insist upon.

It is curious that in Dr. Stone's Croonian Lecture on this subject, as given in the 'Lancet,'<sup>1</sup> neither of the words discord and dissonance is ever used, and no explanation of the tremulous or quavering quality of the sound is seriously attempted. Once only does he come near it, when, referring to the voice sound transmitted through fluid contained in a bag, he says :

"The principal point to be noted was the raising of its pitch. This was usually to the extent of a minor third above the note heard by the open ear, but it seemed also to be accompanied by a squeaking or bleating sound, due

<sup>1</sup> Vol. ii, 1879, p. 189.

to the presence of notes some octaves higher, or too remote for the exact pitch to be ascertained."

Now *squeaking* and *bleating* are different sounds. The squeak of a mouse is a very high but pure note; the bleat of a sheep or goat is discord. The above statement that high or remote harmonics were present will account for a squeak, but does not without further details explain the bleating, which after all is the essential feature of the morbid sound known as ægophony, or goat's voice.

The exception to which I have referred is Dr. Alfred Luton of Reims, the author of the article "Auscultation" in Jaccoud's 'Nouveau Dictionnaire de Médecine et de Chirurgie.' I have quite recently found that in that article, contained in vol. iv, and published in 1866, he states (p. 128) that ægophony "presents some analogy with those phenomena of sound in which the vibrations are discordant or disagreeable to the ear, forming a succession of noises interrupted by very close intervals." In 1880, and again in 1890, he published articles in which he expanded this idea of discord, with some reference to Helmholtz's work on Harmonics and the use of resonators, but he does not seem to me to take the correct view as to the occurrence of discord in this instance. I shall refer to his view again. I may say that it was after I had convinced myself of the relation of discord to ægophony that I first met accidentally with a reference to Luton's work.

We have now to consider what changes in the physical state of the lungs or pleura will bring about the conditions which necessitate discord in the transmitted sound.

We have already seen that a compound tone, such as any word spoken by the human voice is, or may give rise to, discord if it contains very high or loud partials or harmonics,—for this reason, namely, that beats are produced between the harmonics of such frequency per second as to be disagreeable to the human ear.



It is also of course true that discord is produced when two unsuitable notes are sounded ; but it is obviously more satisfactory, in the case of a single word spoken by a single individual, if we can find an explanation that does not call for the simultaneous utterance of two sounds.

In the case, then, of the single sound, discord is favoured by—

1. The pre-existence of high harmonics in considerable force.
2. The reinforcement of those high harmonics.
3. The suppression of the lower harmonics and the fundamental tone.

1. *Existence of high harmonics in considerable force.*—On this point the ground is already clear. As Dr. Stone pointed out, the fact that ægophony is best heard in the case of words or syllables containing the vowels *e* and *i* ("three," "ninety-nine") corresponds with Helmholtz's discovery that these two vowel sounds required a considerable strength of the higher harmonics.

2. *Reinforcement of the higher harmonics ; and*

3. *Suppression of the lower harmonics and fundamental tone.*

It will be seen that these two propositions must be taken together. Let us consider some of the facts of *resonance*. If a number of different tones are sounded simultaneously in the immediate neighbourhood of a tube closed at one end, or other form of resonator, some one particular tone which corresponds in its wave length to the length of the tube will be increased in loudness, in consequence of the air in the tube being set in vibration.

If this resonator is applied to one ear, and the other ear be stopped, the observer will fail to hear, or hear but feebly, the other notes which do not correspond to the resonator.

Now the transmission of the voice to the surface of the chest has long been admitted to be a phenomenon of resonance ; it is called *vocal resonance*. The synonym of

bronchophony is *increased vocal resonance*, and yet the full significance of resonance in the production of ægophony seems not to have been recognised even by Dr. Stone in his discussion of this subject. The voice in the healthy individual is resonated in the bronchial tubes, and the loudness with which the vibrations are heard in the chest is due to their reinforcement in the bronchial tubes. The bronchial tubes, then, are resonators of certain tones: if they are completely obstructed or compressed, there is no resonance at all; the voice is practically inaudible through the chest. However superior solids may be as conductors of sound to liquids and air, the completely solid lung, with compressed bronchi, does not bring the vocal sounds to the surface of the chest. It is, indeed, practically known and admitted that the transmission of the voice sound to the surface of the chest is dependent on patent bronchial tubes, and does not take place, or in the feeblest possible way, if the bronchial tubes are, as tubes, abolished.

Vocal resonance, as is well known, varies much in individuals: it is most marked in males with low-pitched voices; it is feeble in women and children with high-pitched voices. This is not explained by conduction; it is explained by assuming that the bronchial tubes as resonators correspond more nearly to the lower pitched vocal sounds than to the higher pitched vocal sounds.

The application of this to ægophony is the following:— If we have a stopped tube which resonates to a given note, and then shorten the tube by one half, it will not now resonate to the given note, but will resonate to a note of twice the number of vibrations per second, and one octave higher.

If we can substitute for the normal condition of the bronchial tubes a condition which will make them resonate the higher tones, we shall be in the way to produce ægophony; we shall at the same time suppress the lower tones, because there is nothing to resonate them, and we shall reinforce the higher tones because the vibrations of

these higher tones meet with air-columns of lengths which correspond to the length of their undulations. If the higher harmonics of certain tones are sufficiently reinforced, discord results.

I maintain, therefore, that ægophony is produced by such modifications of the bronchial tubes as enable them to resonate the higher tones and harmonics of the vocal sounds, and render it impossible for them to resonate the lower tones.

Admitting, therefore, the justice of Dr. Stone's observation that the lower notes are suppressed, I believe that is not mainly by the resistance of the fluid or the difficulty of transmission from medium to medium, as Dr. Bristowe suggests, but by the fact that they are suppressed for want of suitable resonating spaces on the laryngeal side of the pleura.

I shall support this position by some other considerations, positive and negative.

Is the explanation that the lower tones are cut off by the fluid media consistent with our knowledge of acoustics? Under what law of acoustics does this "filtration" through media come?

Dr. Stone says that ægophony is to be explained as "vocal resonance divested of its lower fundamental tone by the deadening effect of a layer of more or less inelastic fluid."

In reference to this I may say that, on the one hand, I have hitherto failed to find in works on acoustics any statement to the effect that liquids will cut off low tones and transmit high tones; and, on the other hand, I have so far failed to get this result experimentally. I have applied bladders and india-rubber bags of water to the backs of healthy people, and have auscultated through the bag: the voice has always come through with diminished intensity, but without any ægophony or suppression of the lower tones. I fitted an india-rubber bottle containing water against the lower end of a speaking-tube in my house, and applied the ordinary binaural stethoscope; an assistant spoke

down the tube, a vertical distance of about thirty-four feet, and also sounded a pitch-pipe yielding A and C. The sounds were transmitted quite clearly without any ægophony or discord, though not as loudly as they would have been without the intervention of the water-bag.

Dr. Bristowe elaborates this part of the theory somewhat, and rightly excluding all questions of the relative conducting powers of gases, liquids, and solids, from consideration, he points to the difficulty of transmission between different media; and then, as an explanation of the filtration or differential transmission of the tones, he states that "high notes are more penetrating than those of graver tone, and hence would be less likely to suffer in their passage."

But there are difficulties in the way of our accepting this statement. Everyday experience is in direct opposition to it. It is the bass notes of the organ which at the end of the Voluntary vibrate through the seats of the church, and send a thrill up the backs of the congregation; it is the bass notes of the big drum which can be heard at a distance of half a mile, when the fifes and piccolos are inaudible; it is the bass notes of the piano which in their wearisome tum-tum are alone heard through the walls of our next-door neighbours or the next-floor lodgers; and it is the accompaniment only of the street organ, heard through closed windows, which prevents us from settling down to an original article when the said instrument is played in the adjacent street or the mews behind.

In all these instances the bass notes carry further, and have more penetrating power so far as air space, brick walls, doors, and windows are concerned. I cannot recall an everyday experience involving transmission through liquid.

That some high notes should be "penetrating," and that we should talk of "piercing shrieks," referring generally to the higher notes of the female register, is, I think, to be explained by the same laws that I am appeal-

ing to, for an explanation of ægophony, namely, those of resonance. The sounds which are penetrating to our ears are those which resonate in our external meatus,—that is, those whose lengths in air correspond to the length of the human external meatus. If anyone sing up the gamut, he will find two or three consecutive notes which sound much fuller than either those above or those below; they seem to “ring in the ear,”—and, indeed, in more technical terms, they do resonate in the external meatus. But the sounds above them do not resonate in a cavity for which their wave lengths are too small, and those higher notes are not more penetrating than those which are lower, and certainly they are not more penetrating in proportion to the rapidity of their vibration.

The resonating properties of the external meatus are, of course, recognised in works on acoustics, and the cavity is stated to correspond to the wave lengths of tones between  $E^3 = 2560$  vibrations, and  $G^3 = 3072$  vibrations.

In no other sense, I believe, can high notes be shown to be more penetrating than low ones.

In support of the statement of his belief that coarse vibrations are stopped by inelastic fluid, Dr. Stone has drawn a comparison between the properties of light and sound, implying that the greater penetrating powers of the notes of more rapid vibrations of sound is an illustration, on a small scale, of the much greater penetrating powers of the waves of light.

The comparison is unsuitable, as the vibrations take place in different though co-existing media; and it suggests that sound-waves can never penetrate where light is absolutely cut off—a proposition difficult to maintain.

But I shall bring forward in support of the opposite view an illustration from that very subject of light to which reference has been made. The colour of the sky was thus explained by Professor Tyndall. The vibrations of greater rapidity, which cause blue light, are reflected from particles in the atmosphere; while the red rays, which are due to vibrations of less rapidity and large intervals,

are enabled to pass by and overleap, so to say, these obstructions in their way. We see the reflected blue rays ; we lose the transmitted red rays. Here is the recognition that, in the case of vibrations in the same medium, those of which the vibrations are slower and of greater wave length, have greater carrying or penetrating power than those which have greater rapidity and less wave lengths.

Here the analogy is complete, and we see that in each case the slower vibrations carry farther.

Dr. Luton,<sup>1</sup> to whom I have referred above, supposes that the discord is produced between two sounds which take different routes, one through the liquid and the other outside it; and that the former is delayed in its transmission. His most recent statement is as follows :

“ Now we have already said that ægophony results very probably from the superposition of two sounds at least in musical discordance. Of these two components, one is the vocal sound transmitted entirely by the solid parts of the neck, chest, and sometimes even by the indurated lung. The other, more feeble and more distant, has to pass through the liquid pleural effusion, and undergoes refraction thereby. These two sounds, although emanating at the same moment from the same point, are dissociated in consequence of the slight delay which the effused serum imposes on the vibrations which pass through it ; and both arrive simultaneously at the ear, but with a delay sufficient to give rise, by their mingling conflict, to that discordance which is called ægophony.”

There is a want of clearness about this explanation, which imagines tones arriving simultaneously, but one behind the other ; but the main objection is that the hindrance which a layer of liquid opposes to the sound-waves does not result in loss of their frequency per second as waves, but in diminution of their amplitude ; it

<sup>1</sup> Loc. cit., tome iv, p. 128 ; ‘ Union Médicale et Scientifique du Nord-Est,’ Reims, March, 1880, p. 65 ; ‘ Union Médicale du Nord-Est,’ Reims, May, 1890, p. 168.

is not pitch that is affected, but loudness. His subsequent record of success in separating one of the tones or harmonics by means of a resonator, only proves that there is discord from the co-existence of two tones of a certain relation to one another; it does nothing to confirm his view of the manner in which the dissociation arises.

The theory requires also that ægophony should be always heard at the edge of the liquid, which is not necessarily the case, as I hope to show later. We see, then, that transmission through a liquid medium is not sufficient to account either for the suppression of the lower tones, or for the discord of the higher.

I will now call attention to the different familiar noises with which ægophony is compared, and I shall attempt to show that their occurrence has nothing whatever to do with transmission through liquid media, but solely with conditions of resonance. Indeed, it was this comparison which first convinced me that an explanation must be found outside the relation of liquid to the transmission of sound-waves, and also led me to recognise that in the phenomena of beats or discord we must find the cause of the tremulous quality of the sound.

These noises are the bleating of a goat, the human nasal voice, the noise of the Punch-and-Judy show, and the doubtful sort of music made by wrapping some tissue-paper round a comb and then blowing on it. In each case there are notes of high pitch, and tremulous, twanging, discordant quality. The human nasal voice is produced by the resonance of the laryngeal vibrations in the meatuses of the nose, instead of, or in addition to, their being resonated in the cavity of the mouth. The meatuses are shorter and narrower passages than the buccal cavity, and they will resonate high notes, which the mouth cannot; while the mouth will resonate low notes, which the meatuses cannot. If there is little resonance in the mouth, the lower tones will be suppressed; if there is resonance in the nose as well as in the mouth, the higher tones will be at least reinforced to a greater extent than

occurs in the ordinary speaking voice. The instrument which is employed for producing the well-known Punch-and-Judy voice consists of two small strips of tin bent into a gentle curve, and bound to each other with their concave faces opposite, so that they enclose a fusiform space; across this and parallel to the strips runs a piece of tape, dividing the fusiform space longitudinally and equally. The tape can be made to vibrate by blowing between the strips, and produces a discordant sound; the construction of the instrument, which is about an inch and a quarter long and a quarter of an inch wide, obviously provides for the formation and resonance of higher tones. In the comb organ it is obvious that the sound is due to resonance in the spaces between the teeth of the comb. We may probably with safety assume that the bleating of the goat is due to resonance of the laryngeal vibrations in some cavities, whether nasal or buccal, which only correspond to the higher tones; and it is not very likely that it depends upon the presence of a layer of fluid placed anywhere to intercept the lower tones.

I may here, perhaps, deal with the position in which the morbid sound is usually best heard, as this is sometimes thought to have lent some support to the prevailing theory of its production. It is commonly stated that ægophony is best heard in the neighbourhood of the angle of the scapula, or at the upper level of the liquid. Now of these two statements I think the former is the more accurate, but then it is very incomplete; while the latter is quite often contradicted by the facts. It is this latter statement, however, which seems in accordance with the current explanation of ægophony, because a thin layer is thought to be sufficient to modify the voice, while a thicker layer will entirely suppress it.

As a matter of fact, the area over which ægophony is heard may be entirely within the area of dulness, so that the upper margin of the former does not approach the upper margin of the latter; the area of ægophony may reach down to the tenth or eleventh rib, when the dulness



reaches above the angle or even above the spine of the scapula: the area of ægophony may occupy a vertical extent of five or six inches. Analysing twenty-one cases in which I carefully ascertained the limits of ægophony, I find nine in which it occupied a small area of one to five square inches below the lower angle of the scapula, either close to it, and then generally on the outer side; or at some distance below it, and then generally near the spine. In all cases but one ægophony was below, and mostly several inches below, the upper margin of the dulness (Figs. 1—9).

In four cases ægophony occupied a band of about six inches long and two or three broad, running outwards transversely or somewhat obliquely from near the spine to the posterior border of the axilla; twice across the angle of the scapula, and twice entirely below it, but always far below the upper margin of the dulness (Figs. 10—13).

In eight cases ægophony spread over a large area. In seven of these it occupied the base, reaching from above the angle of the scapula nearly to the bottom of the chest, with a vertical extent of four to seven inches, and laterally extending from the spine to the posterior border, or even the anterior border of the axilla (Figs. 14—24). In the eighth case there was an oval area running from the angle of the scapula upwards and forwards into the axilla (Figs. 25, 26). In six out of these eight cases the dulness reached some distance above the upper margin of the ægophony, and it would be quite correct to say of some of them that ægophony occupied the lower two thirds, and of others that it occupied the middle, of the area of dulness. In one of these cases there was for a short time ægophony over the front of the same side laterally from the sternum to the anterior axillary border, and vertically from the third to the fifth rib; it corresponded here to the upper half of the dulness (Fig. 23).

In another of these cases the right chest was dull back and front from apex to base; ægophony was well marked

from the vertebræ to the anterior axillary line, and had a vertical extent of six inches from just above the lower angle of the scapula to near the base. From this chest seven pints of serum were removed by the aspirator (Fig. 14).

From such facts it seems impossible to draw any inference as to a constant relation between the quantity of pleural serum at any one point and the production of ægophony. Probably the most correct description of its position would be this: It occurs most commonly in the lower half of the back within the limits of the area of dulness; if it occupies a small area, it is rather more frequently close to the angle of the scapula than remote from it.

It has now to be shown what is the relation between the liquid which undoubtedly exists in so many cases when ægophony is heard, and the altered conditions of resonance upon which I have attempted to show it depends.

Liquid cannot exist in the pleural cavity without modifying the condition of the lung, which must be to a certain extent compressed; and compression of the lung has been stated by many writers to be the cause of ægophony, but without sufficient explanation of the *modus operandi*. Graham Brown<sup>1</sup> writes, "Most observers are agreed that it depends upon compression and partial obstruction of the bronchi." Certainly, if it is to resonance that we are to look, we must consider the influence of the bronchi; and it seems to me that we can imagine that compression should so modify the size or working length of the bronchi as to alter their relations to particular notes,—to convert them, as it were, from buccal cavities into nasal meatuses, and thus at the same time to suppress the lower harmonics by declining to resonate them, and to reinforce by resonance those harmonics which are highest.

The fact that ægophony is often a transitory phenomenon, and that it is not often heard when the chest is

<sup>1</sup> 'Medical Diagnosis,' Edinburgh, 1882.

full, would then also be explained ; for when the fluid is abundant the bronchi are completely closed, and no resonance in them takes place at all.

According to this view ægophony ought sometimes to be heard in other conditions than pleuritic effusion,—conditions, that is, affecting the bronchi either by compression or some other kind of obstruction. And, indeed, it is believed, as I have already stated, by more than one writer, that such occasions do occur. Dr. Finlayson<sup>1</sup> says, “It is also found in the course of pneumonia and in inflammatory thickening of the pleura.” He adds, “Its cause is still a matter of dispute.” Guttman,<sup>2</sup> although he explains it by the presence of fluid, points out that Skoda recognised it in pneumonia, caseous pneumonic infiltration, and even between the shoulder-blades in perfect health ; and he says that no explanation of its causation under these circumstances has been offered. Fraentzel<sup>3</sup> says, “It is an unimportant variety of bronchophony, and is not a characteristic phenomenon of pleuritic effusion.” Osler<sup>4</sup> also says it may occur in pneumonia. Under the head of the physical signs of that disease he writes, “The bronchophony may have a curious nasal quality, to which the term ægophony has been given.”

But, on the assumption that a modification of the bronchi is competent to produce ægophony, it is not so difficult to imagine how in some of the above conditions the necessary requirements may be brought about. For instance, in pneumonia it is well known that tactile vibration may be entirely abolished without the presence of any fluid. I had an example of the kind a few years ago, in which tactile vibration was absent only a few hours before death over a large part of the back, and *post mortem* the affected lung was unusually distended

<sup>1</sup> ‘Clinical Manual for the Study of Medical Cases,’ 3rd edit., 1891, p. 600.

<sup>2</sup> Loc. cit.

<sup>3</sup> Ziemssen’s ‘Cyclopædia of Medicine,’ English translation, vol. iv, 1876.

<sup>4</sup> ‘Principles and Practice of Medicine,’ Edinburgh, 1892, p. 520.

by the enormous quantity of effused inflammatory products. It may be supposed that in such a case the bronchial tubes are actually obliterated by the pressure of the new products. Another way in which the passage of the air through the tubes may be hindered in pneumonia is by the formation of fibrinous clots in the tubes themselves. If either one of these bring about obliteration of the tubes, either of them might bring about such a modification of the tubes by narrowing or otherwise, as to alter its resonating qualities in the direction of suppression of low tones and reinforcement of high tones.

This possibility is confirmed by the following case which was under my care in 1887. C. G—, æt. 47, was taken ill in November with shivering pains in the left side and epigastrium.

He was admitted on the 5th, when he was very ill with dyspnoea and quick pulse and respiration. When examined at 9 p.m. there was dulness over the lower two thirds of the left chest behind, but none in front. In the lower half of the chest behind was distant bronchial breathing and ægophony. The ægophony extended from the eighth rib to the twelfth, and from the spine to the posterior axillary line. Tactile vocal vibration was very deficient, if not absent. There were no râles. The sputum was viscid and green. (The chest had already been explored by the house-physician without result.)

The following day, at 11.30 a.m., the sputum was less viscid, and of greenish-yellow pink colour; it gave a faint blue reaction with the guaiacum test. The physical signs were much the same, but the whole left back was dull; bronchial breathing was more extensive, but still distant over the lower half. Ægophony was heard again, but its extent was not measured. I concluded that the severity of his symptoms was mainly due to pneumonia, and that even if fluid was present, its removal would not materially improve him. However, at 6.30 p.m., as he was becoming worse, we explored in the eighth and ninth spaces, and drew off only a thin, turbid, grey-green fluid

containing air bubbles, which filled the needle but did not rise into the syringe. He died at 8.30 p.m.

At the post-mortem examination the whole of the left lung was in a condition of grey hepatisation, except one piece the size of an orange, which was hepatised but still red. It broke down readily under the finger, and exuded pus copiously; the upper lobe was congested, and a little condensed where it was in contiguity with the lower; but it still contained air, and was slightly œdematous. It weighed 67 oz. The bronchi contained mucopus, but some of the lower bronchi traversing the consolidated lobe were filled with long plugs or casts of fibrin.

The left pleura was universally adherent by recent pleurisy over the parietal and visceral layers, and there was also interlobar pleurisy.

If ægophony is to be explained by modifications of the tubes, it is obvious that some intra-pulmonary conditions may be supposed capable of producing it, as well as such extra-pulmonary conditions as the presence of pleural liquid. As I have already stated, the evidence of several observers is to the effect that conditions do occasionally arise, independent of fluid, which may produce ægophony. What I have endeavoured to show in this paper is that the presence of pleural liquid, viewed as a medium capable of transmitting, and modifying in that transmission, the sounds of the voice, is not necessary to explain the occurrence of ægophony; and that this morbid sound is merely a discordant modification of the voice, due to altered conditions of resonance in the bronchial tubes. In regard to pleural effusion, it seems to me at present almost a hopeless task to attempt by pathological investigation to find out the exact change in respect of length which the particular tubes undergo, in which this special resonance takes place; that is, to find out the exact difference between the tubes of the small portion of lung, over which ægophony is heard, and those of the lung, under otherwise exactly the same conditions,

over which ægophony is not heard. And it is difficult to invent a satisfactory hypothesis, when we consider the different relations which occur between the amount of the effused fluid and the surface extent of the morbid sound. I shall be only glad if anyone else will devise experiments or propound hypotheses which will effect what is wished. The case of pneumonia I have recorded suggests that it may be more easy in some cases of consolidation, apart from liquid compression, to see how the resonance in the tubes is likely to be modified.

### EXPLANATION OF DIAGRAMS.

FIG. 1.—Female *set.* 16. Ill one week. Signs of pleuritic effusion. Heart displaced to right of sternum. *Ægophony* only faintly near the angle of the scapula. Withdrawn by aspiration the same day, 65 ounces.

FIG. 2.—Male *set.* 42. Ill three weeks. Pneumonia followed by empyema. *Ægophony* near the angle of the scapula. Pus found by exploration in ninth space. Incision.

FIG. 3.—Male *set.* 60, admitted after ten days' illness, with signs of fluid at right base. Exploration on several occasions negative: once fifteen minims serum withdrawn, but aspiration failed. Three weeks after admission dulness of same extent as before; *ægophony* near angle of scapula.

FIG. 4.—Male *set.* 22. Empyema following influenza. *Ægophony* near angle of scapula. Sero-pus found on exploration.

FIG. 5.—Male *set.* 36. Ill five days with obvious pneumonia. Rusty sputum for two days. *Ægophony* at a point on the right side near the spine and midway between the lower angle of scapula and the last rib.

FIG. 6.—Male *set.* about 37. Three weeks ill with pains in joints. Very little complaint as to chest. Physical signs of fluid at right base. Faint *ægophony* below angle of scapula.

FIG. 7.—Male adult. Pneumonia on sixth day; rusty sputum. Bronchial breathing affecting the whole of the right lung, except lower part in front. Faint *ægophony* close to angle of scapula.

FIG. 8.—Male *set.* 45. Influenza a week ago; was a little better, then had pulmonary symptoms. Physical signs of pneumonia, but all sounds rather faint and might be due to fluid. Faint *ægophony* at extreme base near the spine.

FIG. 9.—Male *set.* 60. J. A.—Ill seven weeks with trouble at right base. Right base now dull from tenth rib downwards; no vesicular murmur; faint tactile vibration. *Ægophony* two inches vertical extent and three inches laterally. Needle in tenth space drew clear serum.

FIG. 10.—Male *set.* 28. Ill a week with obvious pneumonia. Sputum rusty at first, now mucous-pus. Dulness to spine of scapula. Breathing obscurely tubular. *Ægophony* from eighth to tenth rib.

FIG. 11.—Male *set.* 40. Signs of fluid over right chest, which was bulged at the lower part, the costal margin being raised. Dulness nearly up to spine of scapula. *Ægophony* in a broad band, just below and including the lower angle of the scapula. Some time later two pints of liquid were withdrawn.

FIG. 12.—Male *set.* 40. Pleurisy left side. Dull all over back on that side. *Ægophony* as shown. Fifty-eight ounces clear serum withdrawn.

FIG. 13.—Male *set.* 49. Right pleuritic effusion and ascites. Dulness up to midway between spine of scapula and lower angle. *Ægophony* along the ninth rib. Three days later one pint of serum withdrawn.

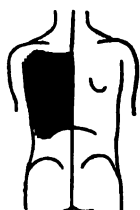


Fig. 1.

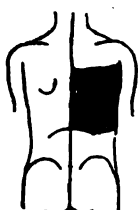


Fig. 2.

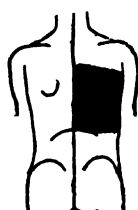


Fig. 3.

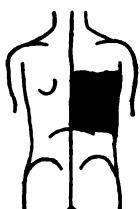


Fig. 4.

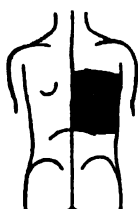


Fig. 5.

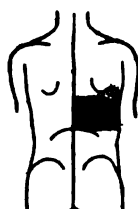


Fig. 6.

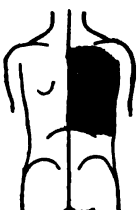


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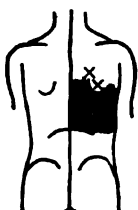


Fig. 8.

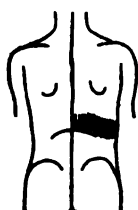


Fig. 9.

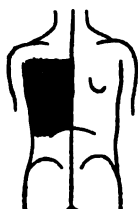


Fig. 10.

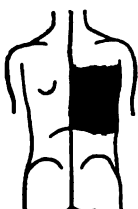


Fig. 11.

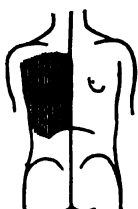


Fig. 12.

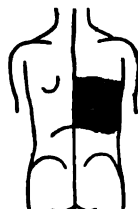


Fig. 13.

Dulness |||||

Bronchial breathing X X

Ægophony /////

Rales ····

EXPLANATION OF DIAGRAMS (*continued*).

FIG. 14.—Male *set*. 34. Right chest uniformly dull. Heart's impulse two inches outside left nipple. *Ægophony* well marked over lower two thirds of back, reaching forward to anterior axillary line; vertical extent in back at least six inches. Seven pints of serum withdrawn.

FIG. 15.—Male *set*. 49. Dull nearly up to spine of scapula: obscure, high-pitched, almost bronchial breathing for  $1\frac{1}{2}$  inches above angle of scapula. *Ægophony* typical and well marked over right base from seventh space inside scapular angle to the eleventh rib, and extending laterally four inches from spine.

FIG. 16.—Male *set*. 60. Ill fourteen days, beginning with hæmoptysis, then febrile. Impaired resonance, not absolute dulness, over left back. Tactile vocal fremitus rather faint. Bronchial breathing up to spine of scapula. Bronchophony over much of back becoming *ægophonic* in character.

FIG. 17.—Same patient five days later. Impaired resonance up to spine of scapula. The voice-sound over the whole of this area has an *ægophonic* twang quite distinctly, but most marked in the axilla at the border of the scapula. Nowhere, however, could it be called loud typical *ægophony*. Exploration yielded no result. Patient ran the course of phthisis and died two months later.

FIG. 18.—Male *set*. 32. Pleuritic effusion. Ill three weeks. Dull over whole of left back. *Ægophony* from spine of scapula to tenth rib, and laterally from spine to posterior axillary line.

FIG. 19.—Male *set*. 13. Empyema. Left chest dull front and back, and apex to base. Heart beating to right of sternum. *Ægophony* over large part of back as shown, extending to axillary line, not in front.

FIG. 20.—Male *set*. 16. Pleural effusion left side. Ill one week. Dulness behind from apex to base and in front from second space downwards. Heart beating to right of sternum. *Ægophony* in lower half of back over an area measuring at least  $6\frac{1}{2}$  inches by 4 inches. Forty ounces of serum withdrawn the following day.

FIG. 21.—Male *set*. 33. Pleurisy. Ill one week. Dulness of left chest to one inch above angle of scapula. *Ægophony* half an inch above and below angle of scapula and near spine on same level.

FIG. 22.—Same patient six days later. Dulness nearly up to spine of scapula. *Ægophony* distinct at upper half of dulness as shown.

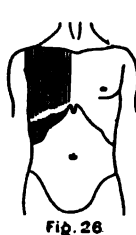
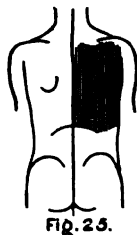
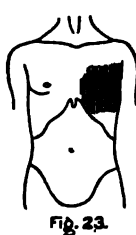
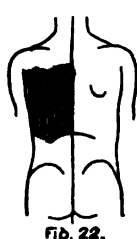
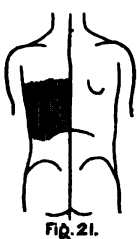
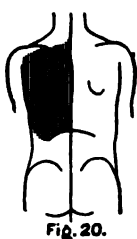
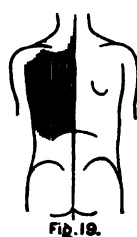
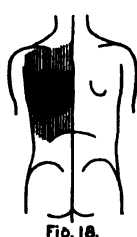
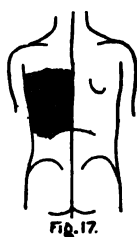
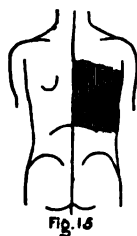
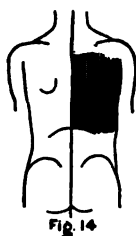
FIG. 23.—Same patient after another four days. *Ægophony* behind unaltered; in front occupies upper part of area of dulness around nipple as shown.

FIG. 24.—Same patient after another five days. Dulness as before. *Ægophony* from midway between spine and angle of scapula for four inches downwards and laterally from spine nearly to border of latissimus dorsi.

FIGS. 25, 26.—Male *set*. 9. Rigors on September 12th, followed by typical pneumonia. Suspensions of fluid on September 19th and 22nd. September 27th, physical signs as shown. *Ægophony* from below and outside angle of scapula outwards, upwards and forwards to axilla and pectoral region. Pus removed by incision the same day.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. vii, p. 61.)





Dulness |||||

Bronchial breathing × ×

Ægophony /////

Râles . . . . .



# ON NERVOUS SYMPTOMS AND MORBID CHANGES IN THE SPINAL CORD

IN

## CERTAIN CASES OF PROFOUND ANÆMIA.

BY

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IN 1887, Professor Lichtheim described at the Congress für innere Medicin<sup>1</sup> in a paper "Ueber Pathologie und Therapie der perniciösen Anämie" three cases which presented the usual clinical symptoms of that disease. But in addition to these symptoms all of them had also signs pointing to an affection of the spinal cord. In the first case these signs were rigidity, weakness, and ataxy in the legs, with paræsthesiæ. The knee-jerks were present but diminished, and the pupils were normal. The second patient had similar symptoms; but in the third the symptoms differed somewhat: there were lancinating

<sup>1</sup> 'Neurologisches Centralblatt,' 1887, p. 236.

pains and the knee-jerk was lost, so that the resemblance clinically, at least as far as spinal symptoms were concerned, was to a case of commencing tabes.

In this last case there was no post-mortem examination. In the other two, however, distinct changes were found in the spinal cords after death. These were almost complete degeneration of the columns of Goll, with similar but slighter affection of the pyramidal tracts. Besides these changes there were small foci of degeneration in other parts of the lateral columns and in the anterior columns. Lichtheim thus early regarded the changes as resulting from a toxic blood condition.

The next communication on this subject was from the same observer, when at Heidelberg<sup>1</sup> in 1889 he described the conditions which his pupil Minnich had found in the spinal cords of patients who had died from pernicious anæmia, but in whom no symptoms of spinal disease were present. In none of the cases examined was the cord absolutely normal. In half the cases there were small sclerotic foci, which on microscopical examination were found to be the result of hæmorrhages. In the other half of the cases there was distinct degeneration in the posterior columns, differing in intensity in the different cords. The degeneration was not restricted to the posterior columns, but was most evident in these. In all of them Clarke's column, Lissauer's tract, and the posterior roots were absolutely normal. The view was again expressed that the condition was the result of a toxic blood-state, and the changes resulting in the nervous system from another blood-state, namely, diabetes, were referred to as analogous. The analogy has been further strengthened by the more recent researches of Williamson, who found degeneration in the posterior columns of the cord in three cases of diabetes mellitus.<sup>2</sup>

A further very exhaustive examination of Lichtheim's cases was carried out by Minnich, and to those previously

<sup>1</sup> 'Neur. Cent.,' 1889, p. 662.

<sup>2</sup> 'Brit. Med. Journ.,' 1894, i, p. 398.

described other cases were added.<sup>1</sup> Six cases were described, in all of which post-mortem examination was obtained. In all of them profound anæmia was present, with retinal hæmorrhages and motor weakness, sometimes in all limbs, sometimes only in the lower. In two the knee-jerk was exaggerated, in one it was described as present, in another diminished, and in two absent. In one in which the knee-jerk was absent there was no reaction in the pupils to light, but they reacted during accommodation. One exhibited no marked signs of spinal affection. Of the others, all except one showed ataxy and complained of subjective sensations, while most had actual sensory impairment in the limbs, and weakness of the bladder. Lightning pains do not seem to have been present in any.

The spinal cords in all the cases showed similar degeneration, chiefly marked in the posterior columns and in one confined to them. In the others there was also degeneration in the lateral columns, and in two also in the anterior columns; while in all the cases the posterior roots, the grey matter and the peripheral nerves were described as free from changes.

Minnich<sup>2</sup> supplemented his work by an examination of the cords in five cases of pernicious anæmia, the subjects of which had not developed symptoms of affection of the cord before death. The examination was undertaken in the hope that the commencing changes in the spinal cord might be recognised. Two sets of changes were found. The first were capillary hæmorrhages with apparently miliary sclerosis resulting from them. The second set of changes was present in three cases, and consisted in slighter coloration of the posterior columns throughout the cord after hardening with bichromate salts, and to some degree of the anterior and lateral columns in certain parts. The cord as a whole was brittle, possibly as a result of its œdematous condition.

<sup>1</sup> 'Zeitschr. f. klin. Med.,' xxi, 1892.

<sup>2</sup> Ibid., xxii, 1893.

Similar changes were found in three cases of chronic jaundice, in one case of leukæmia, and in one of tumour of the inferior vermis of the cerebellum, that is, in the cords of patients who had become much reduced in consequence of prolonged illness. The author regarded it as not impossible that dropsical changes in consequence of some illness might be the starting-point of a true degenerative affection of the spinal cord.

Van Noorden<sup>1</sup> next described a case in which the prominent symptoms were anæmia, paræsthesiæ in the hands and feet, impaired sensibility in the legs, motor weakness in the arms and legs, with spontaneous jerkings in the former, and finally complete inability to walk. The knee-jerks were lost. In the cord there is said to have been complete degeneration of the posterior columns except of a part next the posterior roots, and also in the lateral columns of the cord. The posterior roots were free from change, and there was no shrinking of the cord, such as occurs in cases of tabes.

Eisenlohr<sup>2</sup> also had a patient, a man of fifty-nine, suffering from pernicious anæmia, who developed motor weakness and paræsthesiæ with, later, loss of control over the sphincters. There was no obvious ataxy and no pupil change, but the knee-jerks were lost. He found degeneration in the posterior columns of the cord, and scattered foci also in the lateral columns.

Two cases of this affection have also been published by Nonne.<sup>3</sup> The first was that of a man of forty-eight, suffering from pernicious anæmia, who developed nervous symptoms, numbness and weakness of the lower extremities, ataxy, girdle sensation, and loss of control over the sphincters. There was no pupil change, and the condition of the knee-jerk varied, being at first diminished, then lost, and later again distinctly present. In the second patient, a man of fifty-seven, uncertainty in

<sup>1</sup> 'Charité Annalen,' 1891.

<sup>2</sup> 'Deut. med. Wochenschr.,' 1892.

<sup>3</sup> 'Archiv f. Psych.,' xxv, p. 2, 1893.

walking, occasional girdle sensation, and numbness in the hands and feet had been present for some months before the anæmia was obvious. When this became profound the motor weakness increased, but ataxy was no longer present. There was incontinence of urine, and the knee-jerks, which previously had been diminished, were now increased. The pupil reflex was not affected.

The cords in both cases showed changes in the posterior columns especially, but also in the lateral and anterior. The areas in these two latter columns seemed to be formed to some extent at least by the coalescence of foci of comparatively recent degeneration. The area of the posterior column contiguous to the posterior roots, the posterior roots themselves, and Lissauer's tract were unaffected.

A very interesting case was recently published by Dr. Bowman,<sup>1</sup> being that of a patient who was under the care of Dr. Hughlings Jackson. The patient was a woman of fifty-three, and on admission to hospital she was found to have great weakness but no actual paralysis, and no change in the reflexes. She was very anæmic. She improved very much, and was able after three months' treatment with arsenic to leave the hospital and to go to a convalescent home. She was, however, readmitted after nine months, and she then had been unable to walk for a month, and there had been occasional incontinence of urine. There was great weakness and ataxy in the arms, while the legs were rigid and semi-flexed, and spasmodic jerking in them were painful and frequent. Sensibility in the lower limbs was also impaired. The knee-jerks were active and the pupil reflex was normal. There was marked anæmia; and this gradually became more and more profound, the ataxy in the arms increased, the legs became more rigid and drawn up, and the anæsthesia on them deepened. Numerous retinal hæmorrhages developed, the patient passed into a delirious condition, and she gradually sank and died.

<sup>1</sup> 'Brain,' vol. xvii, 1894, p. 201.

In the cord, changes were found similar to those described in the previous cases referred to, namely, marked degeneration in the posterior columns, with extensive changes as well in the lateral and anterior. The posterior roots and the grey matter were normal, but the blood-vessels in the most degenerated parts were much altered, the walls of the arterioles being greatly thickened and their lumen much reduced—in some instances occluded.

Such are the cases which in the course of my work on this subject have come under my notice as cases in which spinal symptoms and anæmia of the type known as pernicious were recognised as associated. In all the cases I have mentioned a strikingly similar condition of the spinal cord was present. But I have also met in the course of my investigations with certain other facts which show that a similar condition of the cord has been noticed in other cases, whilst its close association with, perhaps dependence on, the anæmia which was also present, had not been recognised. It is mentioned by Nonne, in the paper to which I have referred, that Leichtenstern described in 1884 two cases of what he called *tabes* associated with anæmia. In neither case was there any history or suspicion of syphilis, and no gastric or other crises. In 1891 Dr. James Putnam, of Boston,<sup>1</sup> described “a group of cases of system sclerosis of the spinal cord, associated with diffuse collateral degeneration occurring in enfeebled persons past middle life, and especially in women.” Of such cases he had seen eight, and in four instances had been able to obtain post-mortem examinations. The symptoms generally consisted in a progressive impairment of both sensory and motor functions of all four extremities, associated after a time with general wasting of muscles and emaciation. The fatal cases ran their course in about two years, in one case four years. In three cases marked inco-ordination of movement was present, and in one lancinating pains. In three the knee-jerk was exaggerated and ankle-clonus was present; in the other case

<sup>1</sup> ‘Journal of Nervous and Mental Diseases,’ February, 1891.



the state of this reflex is not mentioned. Of the eight fatal cases, six were women past middle life, and almost all were in a condition of considerable debility. In several, obstinate diarrhoea was a prominent symptom. Several of the patients are also said to have had lead in the urine. Anatomically morbid changes were found in both motor and sensory areas of the cord. In one the medulla was examined and found normal. In the white columns two sets of changes were present, both degenerative,—one of old standing, the other recent and more acute, chiefly around the older degenerated areas. In the grey matter there was disintegration of nerve-cells. Unequivocal signs of acute inflammation were absent. Slight thickening of the pia was present in three cords, and change in the peripheral nerves was found in the one case in which they were examined.

Dr. Putnam also gives a short clinical account of four other cases in which similar symptoms were present. The illness usually began with paræsthesiæ of the extremities, weakness and inco-ordination usually followed, and finally paraplegia and death. In all there was great weakness with emaciation, and in some at least profound anæmia. In two the state of the knee-jerks is not noted, in the other two it was absent.

I think there can be little doubt that these cases described by Dr. Putnam are to be placed in the same category as those to which I have alluded. The gradual onset of symptoms—weakness and paraplegia, the association of those with, in all cases presumably, and in some certainly, a greater or less degree of anæmia, in some profound anæmia, the gradual increase in the weakness leading to complete paraplegia, the variability in the reflexes, in some exaggerated, in some diminished or absent, characterise the histories and clinical condition in both classes of cases. And the actual post-mortem findings, if not identical, present at least very striking resemblances. There is the same wide-spread sclerosis; the same parts of the cord are picked out; there is the same tendency

to limitation to the similarly situated comparatively small areas in the lumbar region. The changes described in the grey matter in most of Dr. Putnam's cases, however, are not present in the others I have referred to; but, on the other hand, in at least one of these the changes were very slight.

Professor Sir T. Grainger Stewart described<sup>1</sup> the case of a patient who had spent most of his life in New Zealand. He had lived freely, but had never been intemperate. In January, 1889, he had noticed some tingling in the tips of the fingers of both hands. This was most marked after exertion, and was aggravated by cold. He also became shaky and unsteady, his knee-jerks were exaggerated, and ankle-clonus was obtained on both sides. Voluntary power was impaired, especially in regard to fine movements; writing had become difficult; and there was a certain awkwardness in his gait. There were no vaso-motor or trophic changes, but the patient was thin and anæmic. His psychical condition was unaltered. He became gradually worse, the anæmia deepened, and there was greater difficulty in walking. Sensibility also became diminished. He spent the winter at Mentone, but was confined to his room nearly all the time on account of severe diarrhœa. On his return he was more anæmic, there was diminished sensibility, and the difficulty in getting about was much increased. The knee-jerks were still exaggerated, and ankle-clonus was present.

Sir T. Stewart referred this case to the same category as Dr. Putnam's, and I would venture to think that both it and Dr. Putnam's cases belong to the same group as those I have just described.

Dr. Bulloch<sup>2</sup> described, under the title "Hyaline Degeneration of the Spinal Cord," a very interesting case, of which the following is a brief summary:—A man of fifty-one, a farm labourer of temperate life, and with nothing of significance in his previous history, was admitted to hos-

<sup>1</sup> 'Brit. Med. Journ.,' June 6th, 1891.

<sup>2</sup> 'Brain,' 1892, p. 411.

pital complaining of shortness of breath and swelling of the feet and face, which had come on about two months previously. He was well nourished but profoundly anæmic, of lemon-yellow colour, with œdema of both feet and ankles, and also of the face. Examination of the blood revealed great paucity of red corpuscles and change in their form and pigmentation; flame-shaped hæmorrhages were present in both retinæ, but no optic neuritis. He was rather awkward and unsteady when he got up, but this was attributed to the great weakness which was present. He also had occasional delusions. He was treated for the anæmia with Fowler's solution, and improved. While at a convalescent home he was the subject of delusions, but was easily managed, and here gained strength, so that he was able to walk to hospital to report himself on more than one occasion. In the third week of his stay he was observed to be ataxic in his gait. Romberg's symptom was present, and there was paresis of the legs, which increased so much as to necessitate his return to hospital. A day or two after admission the paresis of the legs had become complete paralysis. The limbs were flaccid, and the superficial and deep reflexes were abolished. Weakness and inco-ordination now affected the upper limbs, so that he became unable to feed himself. Sensibility was also diminished, he became apathetic and listless, and passed urine and fæces involuntarily. He had several severe attacks of diarrhœa, he became gradually worse both physically and psychically, bedsores formed, and he died of exhaustion.

No appearances indicative of the profound anæmia are mentioned as being visible in the organs. The liver is described as congested and nutmeg-like in appearance. The spinal cord was softer than usual, and between the third and fifth cervical nerves diffuent and altered in contour. On section here a cavity was seen, which at its lower part was more of the nature of a fissure extending deeply into the substance of the cord, so as almost to divide it into two. After being hardened, the cord was

cut, and sections at different levels showed the presence of what is described as "hyaline matter" in various situations. The pia was thickened, and a layer of this hyaline material was under it, and this had, at different levels, replaced the nervous tissue in various parts of the white columns. In the cervical region it was most abundant in the posterior columns, and was present also in the lateral column. It was also abundant in the grey matter, and no cells could be found. In the dorsal region this degeneration was less extensive, being present only in the posterior columns. The parts of these along the margins of the posterior horns were healthy. No cells could be found in the grey matter. In the lumbar region the change was confined to a small triangular area on each side of the posterior median septum. A few ganglion-cells could be seen in the anterior horns. Below the fourth lumbar nerve no degeneration in the posterior columns was recognisable.

The vessels were thickened and distended with blood-corpuscles, and the septa were enlarged and homogeneous.

This case has also striking similarities to the cases already mentioned. There is the profound anæmia, evidently of the pernicious variety, the symptoms of disease of the spinal cord, and the degeneration in certain areas, similar at least in distribution to that found in Minnich's and the other cases. The chief difference is that in this, as in Putnam's cases, degenerative changes in the cells of the grey matter are described, while in Minnich's and the other no such change was present.

I shall now describe two cases which I have observed, for permission to publish which I am indebted to the kindness of Dr. Hughlings Jackson and Dr. Gowers.

CASE 1.—S. A. P., æt. 50, married for twenty-two years, laundress, admitted to the National Hospital for the Paralysed and Epileptic, Queen Square, February 12th, 1891, under the care of Dr. Hughlings Jackson, on account of numbness in the legs and difficulty in walking.

The history was that her present illness began with numbness in the forefinger of the left hand twelve months before admission. A month later there was a tingling sensation along the ulnar border of the left hand. The outer side of the left leg became similarly affected, and afterwards the right arm and leg became numb and stiff. She stated that she had also suffered from slight darting pains in the left knee on several occasions about three months before admission. There was no trouble with the bowels, and no vomiting or gastric disturbance. For eleven months she had had difficulty in walking on account of unsteadiness and weakness. There had also been some weakness in the hands, so that she could not write so well as usual. There had been no impairment of vision. During the nine months before her admission her friends had frequently remarked on the brown discoloration of the skin. For the six months immediately before admission she had been troubled with frequency of mictarition.

Her health previous to the illness for which she sought advice had been good, with the exception of an attack of what is called "congestion of the liver" four years before admission. She made a good recovery from this. She had two children, aged respectively twenty and twenty-one, both healthy. She had had no miscarriages.

There was nothing important or significant in her family history. Her father died at seventy-two, her mother at sixty,—the latter of some liver disease, it is said, and the former from some unknown cause. She had two brothers, who had both died; the cause of death was not known in either case. She had three sisters healthy. There was no history to be obtained of any family disease.

On admission she looked fairly well nourished, but had a peculiar yellowish-brown colour, which suggested a possibility of Addison's disease. Examination of the flexures at the groin and knee, and also of the axillæ, failed, however, to reveal any abnormal degree of pigmentation in these parts. She walked with difficulty and uncertainty, and the weakness seemed to be greater in the left leg than in the right.

There was unsteadiness in standing, which was distinctly increased on covering the eyes. The knee-jerks were present, that on the right side being more active than usual, while that on the left side could only just be elicited. There was no sensory impairment to be made out, except that the sense of position in regard to the left foot seemed to be defective. The special senses were all normal, and no changes were observed in the optic discs or fundi. There was no albumen or sugar in the urine.

During the next few months the patient became gradually weaker, and by the end of the year was unable to stand. The urine also had become albuminous, and there was retention, necessitating the daily use of the catheter. The knee-jerks were very feeble on both sides.

On January 15th, 1892, it was found that sensibility to touch on the legs was much dulled, especially on the outer sides; incontinence of urine was now present, and neither knee-jerk could be obtained.

On January 24th it was observed that in the left upper limb peculiar spontaneous movements were present, consisting in ulnar flexion at the wrist alternating with relaxation. There were also movements of extension of the proximal phalanges, rhythmical in character, associated with irregular wave-like movements affecting the fingers, not simultaneously, but one after another irregularly. There were also slight movements of extension at the elbow synchronous with those at the wrist, and there seemed to be some movement at the shoulder, although no distinct contraction could be seen in the muscles there. The frequency of the wrist movements was about seventy per minute. They were best seen when the limb was slightly supported at the wrist, and tended to become more irregular when the hand was allowed to lie free.

In the right arm there were also constant movements, not of the same character, but more of the nature of coarse tremor. This hand was also "dropped."

These movements gradually decreased in force and extent, but remained present until the middle of June,

—that is, for about five months. By this time the legs had become extremely flexed at both hips and knees, and voluntary movement was entirely abolished. There was marked sensory impairment in the legs. The movements of the arms now gradually ceased, and those limbs became contracted. The contraction of the legs became extreme, bedsores and cystitis developed, pain was very great, and rendered the use of morphia necessary. The patient became extremely anæmic, gradually sank, and died on the 26th November, 1892.

At the necropsy which was made on the following day the body was found much emaciated, and both arms and legs were contracted to an extreme degree at all the joints, and fixed. As regards the viscera generally, it need only be said that all the organs were extremely pale, and the liver fatty. No change was found in the supra-renal capsules. The cord and brain were preserved; and the former, after being hardened in Müller's fluid, was examined at several different levels. The sections were stained by the Weigert-Pal method, and the following conditions observed. In the cervical region (Fig. 1) there was very extensive sclerosis in the anterior, lateral and posterior tracts of the cord. In the direct pyramidal tract on the right side there was, as the figure shows, a considerable part from which the fibres had entirely disappeared. On the left side, in a corresponding position, there was also a patch of sclerosis, but much smaller in extent. In the lateral regions of the cord the change was as nearly as possible symmetrical, and consisted in an almost complete disappearance of fibres in the region of the direct cerebellar and the crossed pyramidal tracts. On each side there was a small zone of comparatively normal nervous tissue on the outer side of the posterior horn. In the posterior region of the cord also there was a zone of fibres on the inner side of the posterior horns on each side. That on the right was more extensive than that on the left. At each side the fibres were abundant near the horn, gradually fading towards the middle line,

and there was a large area on each side of the posterior median fissure practically free from normal fibres.

In the dorsal region the sclerosis, as shown in the figure (Fig. 2), was similar to that just described, both in degree and extent. Both anterior columns were affected, and in one at least the sclerosis was more extensive than the region of the anterior or direct pyramidal tract. In the lateral and posterior columns the sclerosis was similar to that in the cervical region. Here also, both on the outer and inner side of each posterior horn, there was a small marginal zone of fibres which preserved the hæmatoxylin stain.

In the lumbar region (Fig. 3) the sclerosis was much less extensive, and was confined to the lateral and posterior regions. In the former the degenerated area occupied the position of the crossed pyramidal tract on each side. It was almost symmetrical, and on each side there was a considerable zone of healthy fibres on the outer side of each posterior horn. In the posterior region the degenerated fibres were most numerous in the central region on each side, but immediately to each side of the posterior median fissure there was a small area of stained, apparently normal fibres. The inner or central third also of each posterior column had only a few degenerated fibres. The anterior columns presented no change.

The three figures furnish representations of typical sections in the three chief divisions of the cord.

The grey matter in all the different regions seemed to be normal, except that in the cervical region the cells were perhaps wanting in clearness and definiteness of outline. In the lumbar region there was no change from the normal.

CASE 2.—A. W—, æt. 43, admitted to the same institution on 22nd September, 1891, under the care of Dr. Gowers.

In the beginning of 1888 the patient began to suffer from a tired feeling in different joints. In June, 1890,



he accidentally cut off his right index finger. This was very soon followed by very great weakness, accompanied with vomiting and diarrhoea, and a sensation of "pins and needles" in the feet. He was at this time in Canada, and he came to this country, and after a few months he returned well, and 25 lbs. heavier. At this time he was able to walk five or six miles.

Soon after his return, however, his legs began to grow gradually more stiff. In 1891 he suffered from an "attack of piles," with vomiting and diarrhoea. He woke one morning with a dead feeling in his legs, and a sensation of intense coldness in the abdomen. Since that time he had had no control over his legs, and had been quite unable to walk. He had also had a little difficulty in passing his water, but no pains or sickness, or difficulty in breathing. During the fortnight before admission he suffered from a feeling of constriction round his waist.

His health previous to the time alluded to above had been excellent. He had had quinsy twice, but no venereal disease, not even exposure to the possibility of it. His family history was important, because, although he had four sisters living and healthy, his father died of cancer at the age of fifty-six, and a brother at twenty-eight of "poorness of blood."

His condition on admission can be described briefly. He was a tall, spare, and somewhat pale man, with slight ataxy in the arms and considerable in the legs. There was no sensory impairment, and no change in the electrical reactions of the muscles. The knee-jerk could not be elicited, but the pupils were equal, and reacted normally to light and during accommodation. His condition did not improve, and by the beginning of December the anæmia was very obvious. An examination of the blood showed the corpuscles to be 25 per cent., and the hæmoglobin to be 10 per cent. of the normal. There was some reason to doubt the accuracy of the latter observation, because a week later the corpuscles and hæmoglobin were each 20 per cent. of the normal.

By this time there was impaired sensibility both for touches and pricks, below the knee on the inner side, and on the front and inner side of the thigh, specially on the right limb. On the outer side of the leg and thigh sensibility was good. A few days later (December 17th) hæmorrhages were observed in both retinæ, and subcutaneous ones on each heel. The patient gradually sank, and died on December 24th.

At the necropsy, which was carried out on the same day, no obvious morbid condition of the viscera was found except the extreme pallor, and in the liver obvious fatty change. The brain and spinal cord were hardened in Müller's fluid, and the latter was carefully examined in several different regions. In the cervical region (Fig. 4) considerable degeneration was revealed in different parts. In the anterior columns there was well-marked degeneration on each side of the median fissure, more extensive on the left side than on the right. In the lateral columns there was very extensive marginal sclerosis, the change extending inwards, especially in the region of the crossed pyramidal tracts. The degeneration was almost symmetrical on the two sides. In the posterior columns there was very intense sclerosis, especially in the columns of Goll peripherally. Towards the centre the degeneration was less intense, and outwards on the inner side of each posterior horn there was an area of apparently normal tissue.

In the dorsal region (Fig. 5) the sclerosis was well marked in each anterior column, and similar in extent and intensity, less marked but distinct in the lateral columns, especially the pyramidal tract, and very intense in the posterior columns on each side of the median fissure, although immediately to each side of this there was a small area of less degenerated tissue. In the lumbar region (Fig. 6) the sclerosis was much less intense, and was confined to the lateral and posterior columns. In the former there was a mere patch on each side in the pyramidal tract; in the latter more extensive sclerosis, especially on each side

of the posterior median fissure. But as in the dorsal region, so also in the lumbar, there was immediately on each side of the median fissure a line of less degenerated fibres, and on each side posteriorly, internal to each posterior horn also, there was a bounding layer of comparatively undegenerated fibres.

In all these different regions the grey matter showed no change. The cells were numerous, their outline clear, and they showed no detectable alteration from the normal.

From a consideration of all these cases, it seems to me that we have in them no mere accidental association of anæmia and changes in the spinal cord. The anæmia was a prominent feature in all, and the changes in the cord were so similar and so constant that they must be regarded as in some way dependent on a similar cause. A striking illustration of this occurs incidentally in an observation of Leyden's. This author, in the course of a paper on "*Chronische Myelitis u. System-Erkrankungen im Rückenmark*," mentions a case of what he calls chronic myelitis, and figures the sections at different levels of the cord. The case was that of a patient who had been in a railway accident, and came under observation suffering from distinct symptoms of spastic paralysis. This condition remained stationary for several months, and then rapidly grew worse. He became pale, and symptoms of pernicious anæmia with characteristic changes in the blood were developed. He gradually sank and died, and a comparison of the conditions found in his cord with those in my cases and in the others referred to shows an almost identical distribution of sclerosis.

The question now arises, What determines the changes in the spinal cord? Are they the result of anæmia? is the anæmia the result of them? or are they both the result of the same cause? In most of the cases the anæmia seems to have preceded the spinal symptoms. In one of Nonne's, however, the spinal symptoms are expressly stated to have been present before the anæmia, so it seems most

natural to regard the anæmia and the spinal cord changes with their symptoms as both resulting from a common cause. Such a cause we should most naturally look for in some toxic blood condition, having regard especially to the bilateral symmetry of the changes, and to the constancy with which certain tracts were affected and others spared, so that sections at certain levels in one spinal cord are scarcely to be distinguished from sections at the same level in another (see Figs. 3 and 6). But while I believe that this is the chief cause of such changes as I have described, I think that there may be, in addition to this degeneration, other minor changes, probably the result of that tendency to the occurrence of hæmorrhages which is so marked a feature of pernicious anæmia. This tendency to hæmorrhage is most marked in the retina, but it is also manifested elsewhere, and, as I have already indicated, occurred in certain of the cases to which I have alluded ; and more particularly in the cases of pernicious anæmia without symptoms of spinal cord affection, investigated by Minnich, hæmorrhages, or small foci of degeneration which had apparently resulted from hæmorrhage, have been found in the spinal cord. One very peculiar symptom which was present in the first case which I observed, namely, the curious rhythmical persistent movement of the left arm and hand, and to a less degree of the right, may possibly be accounted for by some such change ; and it may have a little interest and perhaps significance if I mention that at the time of their occurrence the movements were spoken of by Dr. Hughlings Jackson as bearing a certain resemblance to those occurring in canine chorea. As is well known, small hæmorrhages and foci of inflammation are found in the cord of the dog in that disease. I think, therefore, that we may have two factors in the production of the cord changes: (1) the degeneration determined by a blood-state ; and (2) degeneration resulting from actual hæmorrhage, and it may be some slight focal inflammation. That a toxic condition of blood may at least be associated with spinal

cord changes, and probably causally, we have other evidence of, even if we do not regard *tabes dorsalis* itself as of this nature. I have already mentioned the analogy which Lichtheim brought forward of diabetes, universally recognised as a condition in which a poison is present in the blood, and which is frequently associated with symptoms of degenerative disease in the nervous system. When Lichtheim wrote, Williamson's researches were not yet published, but it is now proved by those that sclerosis in the posterior columns of the cord may be associated with diabetes mellitus, and is probably a result of the blood-state. We also have in pellagra a disease probably the result of a chemical poison produced in damaged maize, and characterised by sclerosis of the cord, which offers many points of similarity to the condition to which I have directed attention.

I think, therefore, that it is evident that in certain cases we have a condition of profound anæmia associated with spinal symptoms; and that these symptoms vary in different cases according to the relative amount and intensity of the sclerosis affecting different parts of the cord: that, for example, when the posterior columns are mostly affected we have ataxic symptoms with loss of the knee-jerk predominant, whereas if the changes are most intense in the lateral columns the clinical symptoms more closely resemble those of spastic paraplegia. But it is striking that in the great majority of cases ataxy is so constantly noted, and that, correspondingly, the most constant change in the cord is one of the posterior columns. It remains for future observation to show what relation this condition may have to the group of cases which Dr. Gowers has described under the name of ataxic paraplegia, and to certain others which have hitherto been regarded as cases of chronic myelitis.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. vii, p. 117.)

## DESCRIPTION OF PLATES II AND III.

On Nervous Symptoms and Morbid Changes in the Spinal Cord in Certain Cases of Profound Anæmia (Dr. JAMES TAYLOR).

### PLATE II.

Representations of sections of the spinal cord at different levels in Case 1.

FIG. 1 shows the sclerosis as it affected the anterior, lateral, and posterior columns in the cervical region.

FIG. 2 shows the sclerosis similar in distribution in the dorsal region.

FIG. 3 illustrates the much less extensive sclerosis in the lumbar region, confined to the lateral and posterior columns.

### PLATE III.

Illustrations of sclerosis in different regions of the cord in Case 2.

FIG. 4 shows the distribution of the sclerosis in the cervical region.

FIG. 5 shows the distribution of the sclerosis in the dorsal region.

FIG. 6 shows the distribution of the sclerosis in the lumbar region.

Comparison of Fig. 6 (Plate III) and Fig. 3 (Plate II) indicates an almost identical distribution of sclerosis in the two cases.



Fig. 1.



Fig. 2.

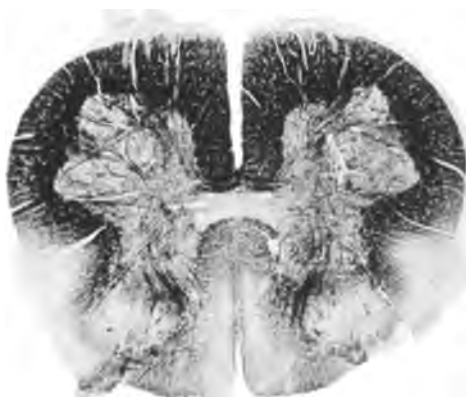


Fig. 3.







Fig. 4.



Fig. 5.



Fig. 6.



A CASE  
OF  
LARGE PELVIC HYDATID  
SUCCESSFULLY TREATED BY PERINEAL  
INCISION AND DRAINAGE  
WITH OBSERVATIONS

BY  
REGINALD HARRISON, F.R.C.S.

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Received February 7th—Read April 9th, 1895.

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THE following case presents some points of interest in connection more particularly with the treatment of pelvic hydatids in the male.

A well-developed man, æt. 45, and above the average stature, came under my observation, at the suggestion of Dr. G. B. Batten, in 1893, with the following history:—He had always enjoyed good health until the commencement of the present illness. In March, 1885, when resident in Australia, where he had been engaged in business for some years, he suffered from retention of urine for the first time in his life. This occurred without the usual history of a urethral stricture, and apparently without any obvious cause. A silver catheter was introduced with

difficulty and pain. It was retained for some hours and then withdrawn, considerably bent, as no urine escaped by it. After the catheter was removed, micturition was spontaneously performed, but with much interruption.

Commencing in this way the subsequent symptoms of the patient's long and continued illness may be briefly stated as those of loss of power lasting for some weeks in the left leg, and the greatest difficulty in voiding both urine and fæces. The pain and distress in this way occasioned quite unfitted him for business pursuits, and gradually led him into the habit of daily taking very large quantities of morphia. His symptoms resisting all kinds of treatment, he returned to England in 1893, being advised before he left the colony to have his colon opened, to relieve the suffering that his constantly-obstructed bowels occasioned.

On his arrival in this country Dr. Batten, in sending the patient to me, wrote "that the pelvis was occupied by a large rounded swelling, which squeezed the rectum flat, and seemed to be a cystic tumour with thick walls," adding that he thought "something besides colotomy might be done to relieve the patient, and enable him to pass water and motions without the frightful agony which he suffers."

On examination, I found the abdomen partially occupied by a tumour, about as large as a fully-developed gravid uterus, situated rather to the left of the median line, and evidently springing from within the area of the pelvis. It was extremely tense though uniform to the touch. On exploring with the finger in the rectum, which was a painful process, the prostate was obscured by a firm tumour, which, as already stated, completely compressed the bowel. On introducing a silver catheter into the urethra, it was found impossible to carry it further than the prostate, but on substituting a long and very flexible instrument, the bladder was easily entered. It was obvious that the latter was pressed over towards the right side, so as to form a tolerably sharp angle with

the deep urethra. It was also apparent that there was no stricture in the ordinary acceptation of the term. The urine was scanty and high-coloured but otherwise healthy. As the patient stated that his fæces at times presented not only a flattened appearance, but were mixed with some viscid or gum-like material, they were carefully examined, with the result that the characteristic hooklets of hydatids were readily discovered. This recognition placed the precise diagnosis as to the nature of the cyst beyond all doubt. The great and prolonged difficulty under which both urine and fæces had been voided, taken in conjunction with the physical state of the abdomen, rendered it probable that not only were the intestines largely distended and impacted, but that the ureters and kidneys were in a similar condition. In fact, on the right side what I believe was a hydronephrotic kidney could be felt. The conclusion arrived at was that the distension was caused by an hydatid cyst, situated between the rectum and the bladder and below the reflection of the peritoneum which forms the recto-vesical pouch.

The patient remained under observation for some time, but was not at first disposed to submit to any operation; his symptoms, however, increasing, if possible, in intensity, he expressed himself as willing to acquiesce in whatever was advised. Mr. Arthur Durham kindly saw the patient in consultation with Mr. Rand (Dr. Batten's partner) and myself, and concurred in the diagnosis that had been arrived at. The nature of the treatment to be employed was very carefully and fully considered, and relative to such measures as had been previously adopted in apparently similar cases. It was decided to open the cyst from the perineum, and to drain from this position. This conclusion was arrived at partly for the reason that it seemed somewhat hazardous to open a cyst which clearly had some slight connection with the intestinal track by any form of abdominal section, where the viscera generally were in a state of extreme distension and im-

paction, and partly because, from the long existence and size of the tumour, it appeared desirable to secure the evisceration and drainage of the cyst at its most dependent point.

The patient, on being anæsthetised, was placed in the ordinary perineal lithotomy position. To indicate the line of the urethra a flexible bougie was passed and retained in the bladder, as, for the reason already stated, a metal instrument would not enter. In order to give me sufficient space to manipulate in a somewhat deep perineum, I made the usual external incision as for lateral lithotomy, without, of course, opening either the urethra or rectum. I then made my way carefully with the finger and knife, between the bladder in front and the rectum behind, to the base of the cyst and exposed it. The latter was first punctured with a large trocar and cannula, but as nothing escaped I freely extended the puncture wound with a probe-pointed bistoury and passed my finger within the cyst. The withdrawal of my finger led to the discharge of a large quantity of fluid and innumerable daughter cysts. The odour was most offensive. The clearance of the interior of the cyst was greatly aided by abdominal pressure exercised by Mr. F. Durham, who, in conjunction with his brother, Mr. Rand, and Mr. W. Braine, kindly assisted me during the operation. In this way, and by the use of forceps and a lithotomy scoop, the cyst was cleared of about half a gallon of contents. There was no hæmorrhage, and the operation was completed by washing out the sac as thoroughly as possible and fixing in a large drainage-tube.

The patient suffered a good deal of cramp in both legs after the operation. Large quantities of both urine and fæces were voided, though, as the bowels seemed to have temporarily lost all power, the discharge from the latter was assisted by enemas. In three weeks the patient was able to leave the surgical home and return to his residence, where his treatment was followed up by Mr. Rand and

Dr. Batten. The sac was carefully drained and washed out; and by attention to the carrying out of this the success of the case was insured. In the course of the after-treatment a considerable number of daughter cysts, in various stages of disorganisation, were discharged by the wound.

The operation was not followed by anything remarkable in the temperature chart, or by what has been referred to by some as an "hydatid rash." His long illness, extending over nine years, combined with the habitual use of morphia in large doses, rendered convalescence somewhat protracted. He called to see me four months after the operation, when he was in excellent health, the power both of urination and defæcation having gradually returned. There was an entire absence of any abdominal or pelvic tumefaction.

Two cases, very similar to the one I have narrated, have recently been published by Dr. Sawkins,<sup>1</sup> of Sydney, which I will briefly refer to. The first was that of a man who had retention and overflow of urine, though the bladder was only found, by the catheter as well as by suprapubic aspiration, to contain a little over four ounces of urine. He also had no rectal control. A tumour was detected by examination of the rectum, which was believed to be a greatly enlarged prostate. The patient further complained of inability to walk and pain along the course of the left sciatic nerve. He gradually sank, and died without operative treatment. An autopsy showed the existence of a large hydatid cyst filling up the pelvis, and pressing on the urethra and bowel. It was attached in front to the base of the bladder, which it invaginated, and behind to the cavity of the sacrum and wall of the rectum. The cyst itself was loosely adherent to the bladder, being directly in contact with the muscular layer. It was covered on its upper surface by the peritoneum of the recto-vesical pouch. There were numerous small cysts attached to pelvic viscera, and in the liver and omentum.

<sup>1</sup> 'The Australasian Medical Gazette,' November, 1898.

In the second case recorded by Dr. Sawkins, in a man aged thirty-nine, the early history was that of retention of urine without previous evidence either of gonorrhœa or stricture. Examination by the rectum gave similar signs of a large cystic pelvic tumour, which, by an exploring needle, was demonstrated to be an hydatid. A suprapubic exploration was made, but as it was found impossible to reach the cyst, the incision was prolonged upwards, and the peritoneum opened. The contents of the sac and the endocyst having been removed, the opening into the envelope was stitched to the abdominal walls, and a drainage-tube inserted. The patient made an uninterrupted recovery in thirty-eight days.

Commenting upon the mode in which these tumours originate in this particular position, Dr. Sawkins, on the authority of the late Dr. Fagge,<sup>1</sup> states that they arise from the growth of an embryo, which has fallen by its own weight into the pelvic cavity after perforating the walls of the stomach. This explanation was also advanced by Dr. John Hunter,<sup>2</sup> who, it is interesting here to note, described the dissection of a case where, in a man, a pelvic hydatid proved fatal by causing retention of urine from pressure on the neck of the bladder.

Mr. Targett, however, in his lectures at the Royal College of Surgeons,<sup>3</sup> takes a different view as to the origin of these tumours, which, Dr. Sawkins states, one of his cases substantiates. Mr. Targett observes: "An examination of all the specimens in the London museums has shown that these cysts were originally placed between the muscular coat and the sheath of recto-vesical fascia. This fascia, besides enclosing the prostate and lower part of the bladder, forms a special sheath for the vesiculæ and vasa deferentia; hence the gradual enlargement of the cyst separates the fascia from the bladder, and thus detaches

<sup>1</sup> 'Principles and Practice of Medicine,' 1st edit., vol. ii.

<sup>2</sup> 'Transactions of a Society for the Improvement of Medical and Surgical Knowledge,' vol. i, London, 1793.

<sup>3</sup> 'Brit. Med. Journ.,' July 29th, 1893.



the vasa and vesiculæ, which henceforth may become incorporated in the wall of the cyst." Consequently, we can no longer regard these cysts as formed within the peritoneal cavity, but as deposited from the large venous plexuses about the base of the bladder, or by the burrowing of the embryo through the mucous and muscular coats. Most of these cysts are of secondary origin, some being furnished by the liver, whilst others, descending from the kidney along the urinary tract, make their way into the cellular intervals in relation with the most dependent portion of the bladder wall. Some of these points are displayed in specimens which I have brought from the Museum of the Royal College of Surgeons.

I will now briefly refer to recorded cases bearing upon the treatment these retro-vesical hydatids have received. In the first place it is not unlikely that a natural cure has on some occasions taken place by the discharge of the contents of the cyst into the intestinal canal, and their escape in this way. In fact, Dr. Hunter, in the case I have referred to, and in the days before abdominal surgery was developed, observes: "When the presence of hydatids is suspected, it would appear to be of great consequence to procure them an outlet; but it must be obvious that, being generally seated in the abdomen, that business must be left entirely to nature." This probably happened, but to a very limited extent, in the instance I have narrated, as hooklets were found mixed with the fæces. The operative proceedings which have been adopted in this class of cases seem to have been of four kinds at least.

(1) Laparotomy, and the opening and draining of the hydatid sac, by attaching the latter to the abdominal parietes. This has already been illustrated in the case recorded by Dr. Sawkins.

(2) By puncture of the cyst through the rectum. Dr. Fairbank<sup>1</sup> records a successful case of this kind. The patient was seen ten years afterwards in good health and

<sup>1</sup> 'Trans. Clin. Soc.,' vol. xxiii.

without recurrence. Tapping or aspirating an hydatid, though a simple operation, is by no means free from risk. Instances of a fatal and rapid form of septicæmia, which at one time led to the impression that under certain circumstances the hydatid fluid was of a poisonous nature, are not uncommon. This danger connected with simple paracentesis is frequently referred to in Australian literature.

(3) By a perineal operation. Mr. Bryant<sup>1</sup> has recorded an instance of a man aged fifty, where the circumstances connected with a pelvic hydatid were not very unlike the case which forms the basis of this paper. The symptoms of both retained urine as well as fæces were pressing and serious, and were evidently associated with a very large pelvic cyst. Puncture from the rectum in three different directions failing to give the necessary relief, Mr. Bryant cut down upon the tumour from the perineum. The cyst was exposed and opened in this way, and, to give more space for the withdrawal of the contents of the sac, the incision was carried backwards into the rectum. In this way upwards of three quarts of hydatid cyst were evacuated. Though the operation was successful in affording relief, granular disease of the kidneys proved fatal on the ninth day after the operation. It seems little short of a miracle that I had not also this serious complication to contend with in my patient's case, after his nine years' severe suffering from obstructed micturition. Fortunately, I believe the kidneys were uninvolved further than, in conjunction with the ureters, having undergone some ascending dilatation.

(4) Mr. Bond,<sup>2</sup> of Leicester, has advocated the opening of these cysts (I presume from whatever position they are most accessible), the withdrawal of the endocyst, and the immediate closure of the envelope without resorting to continuous drainage. Still more recently he has further illustrated the success of this practice. Though it is a method of treatment which has been referred to by Dr.

<sup>1</sup> 'Trans. Path. Soc.,' vol. xvii.

<sup>2</sup> 'Brit. Med. Journ.,' April 11th, 1891, and January 26th, 1895.

William Gardner, of Melbourne,<sup>1</sup> who has had a large experience in these cases, as the "ideal method for living cysts of the kidney," I hardly think it would have been applicable in the case I have narrated. The large extent and probable ramifications of the sac, its connection with the intestinal track, and the subsequent discharge of embryonic hydatids, in spite of the free irrigation, scraping, and cleaning that was practised at the time of the operation, all tend to indicate that continuous drainage was a necessary part of the proceeding. In the case of hepatic hydatids Mr. Bond's practice seems to have been most successful.

In conclusion, it appears to me, having regard to the nature, extent, and attachments of these pelvic parasites, as well as to the urgent and serious manner in which they obstruct both the discharge of urine and fæces, that the perineal method of dealing with them, followed by drainage, has much to recommend it. Even supposing, as it sometimes happens, that these cases are not confined to a single or bilobed pelvic cyst, but are complicated with others of visceral connection—as from the liver, the omentum, or elsewhere—the disposal of the pelvic cyst in the manner I have advocated would tend to remove the chief sources of difficulty to be apprehended in subsequently proceeding with a laparotomy, should further abdominal developments render this necessary.

<sup>1</sup> 'Australian Med. Journ.,' August, 1894; and 'Intercolonial Quarterly Journ.,' August, 1894.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' New Series, vol. vii, p. 126.)



ON THE PROBABLE PATHOLOGICAL IDENTITY  
OF THE VARIOUS FORMS OF  
ACUTE SEPTIC INFLAMMATIONS OF  
THE THROAT AND NECK,  
HITHERTO DESCRIBED AS  
ACUTE OEDEMA OF THE LARYNX, OEDEMATOUS LARYNGITIS,  
ERYSIPELAS OF THE PHARYNX AND LARYNX, PHLEGMON  
OF THE PHARYNX AND LARYNX, AND ANGINA  
LUDOVICI.

BY  
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It is with considerable hesitation that I venture to submit the following paper to the judgment of the Royal Medical and Chirurgical Society. Its thesis—namely, that the various forms of acute septic inflammations of the throat and neck hitherto described under the names of acute oedema of the larynx, oedematous laryngitis, phlegmon of the pharynx and larynx, erysipelas of the pharynx and larynx, and angina Ludovici, are in all probability pathologically identical, and merely represent different degrees of severity of one and the same septic process due to invasion of the throat and neck by various pathogenic micro-organisms—is far-reaching, and nobody

could be more fully conscious than I am, that it can at the present moment only be finally proved by a harmonious combination of clinical, pathological, and bacteriological evidence.

Now I have at the very outset of my remarks to confess that my material, although, I think, singularly complete from the clinical point of view, is somewhat deficient from the aspect of pathological anatomy, and, furthermore, that my bacteriological evidence, so far as my *personal* experience goes, unfortunately is conspicuous by its absence. Under these circumstances, the question why I had not waited, before venturing to submit my ideas to this Society, until I had completed my proofs from every point of view, is so obvious that I feel anxious to answer it somewhat fully before entering upon my subject proper, the more so as I thereby may fairly hope to disarm to some extent hostile criticism.

In the first place, then, let me say that the affections of which the following paper treats, particularly their more serious forms, are, fortunately, so rare that even with exceptional opportunities no single observer is likely to ever command a really large material, and that often even in an extensive specialistic practice years pass without a single case of that nature coming under observation. As a matter of fact I have in the whole course of nearly twenty years' specialistic hospital and private practice, altogether only observed fourteen cases of acute infective inflammations of the pharynx and larynx. Yet this material, small as it may seem to anybody who has not studied the literature of the subject, is, I believe, an exceptionally large one so far as the experience of a single observer is concerned.

Further, I may fairly claim that I have taken to heart Horace's words, "*Nonum prematur in annum*," and have not rushed into print with undue haste. The very first severe case of this sort which I ever observed (this was in 1882, *i. e.* fully thirteen years ago) at once raised the question in my mind whether the present classification of the various forms of acute septic inflammations of the organs of

the throat and neck was scientifically tenable. In every subsequent case which came under my notice I have tried hard to get a bacteriological investigation, but in one single case only, the last one that came under my observation, this opportunity was given to me. I had to wait eleven years for it, and then the evidence obtained from it was unfortunately purely negative. Two years have passed since without a new case of the kind having come under my observation. Under such circumstances it is obvious that it is no good waiting any longer. Possibly again many years may pass before I have the chance of observing another case of the kind, and even then, supposing that I were fortunate enough to get the opportunity of a bacteriological examination, its evidence might again be negative; whilst numbers of such cases may meanwhile pass through the hands of other observers, to whom it might not occur to study the interesting question to which I wish to draw attention unless some incitement were given thereto.

But, apart from these more negative arguments, I have some positive reasons to adduce which seem to justify my present undertaking. My clinical material, although small in numbers, has, as mentioned before, by good fortune been so singularly complete, that from the clinical point of view alone I hope I shall be able to prove that my ideas on this whole subject are more than mere idle speculations.

Again, whilst my own bacteriological investigations have failed, another observer, Dr. Jordan, of Heidelberg, has been more fortunate, and I shall be able to show, in the conclusion of this paper, from quotations of his results and statements, that my ideas on the subject in reality represent an application of the present general views of bacteriological authorities to the special forms of septic inflammations of the throat and neck.

And, finally, I may be permitted to say, that whilst my own inclinations entirely go towards establishing, if possible, new views on the basis of well-ascertained and

incontrovertible *facts*, yet the value of what Professor Huxley has well called a "working hypothesis" ought not to be underrated. I venture to hope that the ideas on the probable identity of the various acute infectious inflammations of the throat and neck, which I shall have the honour to submit to the Society, may be thoroughly tested by subsequent observers, and that their correctness may be even more fully established than I am able to do with the material now at my disposal.

Having thus, I hope, justified my undertaking, I will at once go *in medias res*, and begin by showing from a few recent utterances of competent observers, taken almost at random, the confusion which at present exists in the nomenclature of the various forms of acute septic inflammation of the throat and neck.

Von Ziemssen<sup>1</sup> adopts Bouillaud's terminology in calling "laryngitis phlegmonosa" all those laryngeal inflammations which mainly concern the submucous connective tissue, without, however, being exclusively limited to this. The sub-headings of his chapter are "Œdema Laryngis. Abscessus Laryngis," and he adds that, in order to "simplify" his description, he had admitted into it those forms as well of laryngeal œdema which were of non-inflammatory origin. Hence we find in the paragraph dealing with the etiology such heterogeneous causes as inflammation of the mucous membrane, diphtheria, chemical and thermic irritation, burns and scalds, foreign bodies, mechanical insults, perichondritis, acute infectious diseases (pyæmia, septicæmia, ulcerative endocarditis, typhus, smallpox, scarlatina, measles, erysipelas), again acute and chronic nephritis, malarial cachexia, amyloid degeneration of the kidneys, cardiac affections, emphysema and cirrhosis of the lungs, compression of the vessels of the neck by tumours of the thyroid gland, enlargement of the lymphatic and salivary glands, tumours of the neck, aneurism of the aorta, &c.

<sup>1</sup> 'Cyclopædia of Special Pathology and Therapy,' 1876, vol. iv, 1, pp. 311 *et seq.*



Sir Morell Mackenzie<sup>1</sup> adopts a similar arrangement. He, however, gives his chapter in question the title of "Œdematous Laryngitis" with the synonyms "Laryngitis phlegmonosa. Laryngitis submucosa purulenta," and completely separates from it the erysipelatous form, which is described in a separate chapter as "Erysipelas of the Pharynx and Larynx." The division into inflammatory and non-inflammatory forms of laryngeal œdema is even less distinctly drawn in Sir Morell's work than in von Ziemssen's chapter, but he deserves great credit for having first of all expressed his belief<sup>2</sup> that in nearly all the instances of so-called "simple inflammation" the disease is due to blood-poisoning.

A very different division is adopted by J. Solis-Cohen.<sup>3</sup> This author devotes a separate chapter to each—"Phlegmonous Laryngitis," "Erysipelatous Laryngitis," and "Œdema of the Larynx," and expressly states that phlegmonous laryngitis, though often attended by phenomena of œdema, is to be discriminated on the one hand from serous œdema of the larynx, and on the other from erysipelatous laryngitis, with which it was not unfrequently confounded.

In strict opposition, again, to this last statement, we find Massei,<sup>4</sup> in a very interesting monograph on "Primary Erysipelas of the Larynx," which has exercised considerable influence upon the present views in this whole question, expressing his opinion as follows (l. c., p. 29):—"My own belief I may summarise to the effect that erysipelas of the larynx frequently is nothing else than the process called "laryngitis phlegmonosa," that this disease may occur in a sporadic as well as in an epidemic form, and that it represents a primary, independent, therapeutically not invincible process." In view of this unequivocal statement

<sup>1</sup> 'Diseases of the Throat and Nose,' 1880, vol. i, pp. 272 and 195.

<sup>2</sup> Ibid., p. 274.

<sup>3</sup> 'Diseases of the Throat and Nasal Passages,' 1879, pp. 433 *et seq.*

<sup>4</sup> "Ueber das primäre Erysipel des Kehlkopfs," 1886; 'Deutsche Uebersetzung von Vincenz Meyer,' p. 29.

it is rather bewildering to find in the same author's text-book,<sup>1</sup> not only subdivisions made into "Cedema," "Epiglottitis," "Laryngitis phlegmonosa," and erysipelalous forms, but the latter again subdivided into "Laryngitis erysipelatososa" and "Primary Erysipelas of the Larynx."

Matters, however, became even more complicated when, in 1888, Senator<sup>2</sup> described before the Medical Society of Berlin what in his introductory remarks he called a "little known," and in the conclusion of his paper boldly a "new"<sup>3</sup> disease. To this he gave the name of "Acute infectious phlegmon of the pharynx," and characterised it as "a diffuse purulent inflammation in the deeper tissues of the pharyngeal mucous membrane, which thence is propagated to the larynx and the glands, and secondarily invades also other organs. The disease has attacked (viz. in the author's experience) persons previously in full health, and without any etiological factor being demonstrable."

The reading of Senator's paper was followed by an animated and very instructive discussion,<sup>4</sup> in the course of which the late Paul Guttman strongly contested the view that the disease in question was a new one, and maintained that the cases which Senator had brought forward in support of his statements could be well interpreted as belonging to the class of erysipelalous inflammations of the

<sup>1</sup> "Pathologie und Therapie des Rachens, der Nasenhöhlen und des Kehlkopfs," 'Deutsche Uebersetzung von Emanuel Fink,' 1892, vol. ii, pp. 200—211, 237, 241.

<sup>2</sup> 'Berliner klinische Wochenschrift,' 1888, No. 5, 30th January.

<sup>3</sup> British observers will of course be reminded by Professor Senator's description, of the late Dr. Carrington's and Dr. Hale White's cases of "Phlegmonous pharyngitis" ('Trans. of the Clinical Society of London,' vol. xviii, 1885, p. 164), which are simply prototypes of the affection described by Professor Senator. In the discussion which followed their communication in the Clinical Society ('Brit. Med. Journ.,' 1885, vol. i, p. 382) I for the first time stated the views which have gradually ripened into the present paper, and I have since twice referred to the subject, once in 1890 in the discussion which took place on the question in the Section of Laryngology of the Tenth International Congress at Berlin, and once in the 'Internat. Centralblatt für Laryngologie,' 1893, vol. x, p. 55.

<sup>4</sup> 'Berliner klinische Wochenschrift,' 1888, No. 6, p. 112, *et seq.*

upper air-passages. Virchow declared that on the whole he agreed with Guttman's views, and that he, too, could not exactly define the mutual relationship of erysipelatos and diffuse phlegmonous affections. Sonnenburg stated that such phlegmonous affections as those described by Senator had long been known to surgeons. Senator himself, however, stoutly maintained that the disease described by him had nothing whatever to do with erysipelas, and was also essentially different from the forms of phlegmon mentioned by Sonnenburg.

Just as Massei's monograph had been followed by a host of publications on erysipelas of the larynx, thus now Senator's communication became the starting-point of quite a literature on acute infectious phlegmon of the pharynx, and thus the acute cedematous and phlegmonous inflammation of the upper air-passages, which only twelve years earlier had been described as more or less belonging together by v. Ziemssen, were now split up into (1) idiopathic or essential inflammatory cedema of the larynx; (2) erysipelas of the larynx; (3) acute infectious phlegmon of the pharynx and larynx; (4) phlegmonous pharyngitis from other causes—not to speak of such forms as Strübing's angio-neurotic cedema, and the inflammatory cedema occasionally caused by some drugs, such as iodine preparations, which had also meanwhile been described. Most text-books of laryngology published since then, and the discussion on acute infectious inflammation of the pharynx and larynx which took place in the Section of Laryngology of the Tenth International Medical Congress<sup>1</sup> (Berlin, 1890), vividly reflect the uncertainty and confusion thus produced; it is, however, unnecessary to quote further opinions, first, because I think that the illustrations so far given suffice to show the present unsatisfactory state of the question, and, secondly, because quite recently a very full historical and critical retrospect of the whole matter has been given by Dr. A. Kuttner of

<sup>1</sup> 'Verhandlungen des Xten Internationalen Medicinischen Congresses, Berlin,' 1890, vol. iv, Abtheilung xii, p. 173, *et seq.*

Berlin in an excellent monograph, 'Laryngeal Edema and Submucous Laryngitis,'<sup>1</sup> which has also been published in somewhat abbreviated form in Virchow's 'Archiv.'<sup>2</sup> Dr. Kuttner's views on the whole question are, I am glad to say, so very similar to my own that I could only repeat his description in other words, and I therefore venture to refer those specially interested in the subject to his paper.

My own attention was prominently drawn to the question by a case which I observed in 1882, and which will be referred to further on as Case 14. Although at the time I described it under the title "A Case of Erysipelas of the Larynx, Pyæmia, Death,"<sup>3</sup> I was fully aware, as shown in my concluding remarks, that it was impossible to decide whether the disease started in the cellular tissue of the neck, in the tonsils, or primarily in the larynx. Nor did I conceal the fact from myself that the case might have been regarded, especially if not seen at its very onset, very justifiably by other observers as an instance of Ludwig's angina, the dyspnoea being considered as a mere result of the infiltration of the cellular tissues of the neck, and the laryngeal oedema, if ascertained at all by means of the laryngoscope, being looked upon as simply due to stasis. Finally, I also clearly saw that if a pathological anatomist had performed the post-mortem examination without any knowledge of the clinical course of the illness, his diagnosis might easily have been that of deep-seated phlegmon of the neck with metastasis in several joints.

Anyhow I learned from this case how difficult it sometimes was, in spite of the definite classification of acute septic inflammations of the throat and neck then existing, to accurately define in a given case, such as the one described by me, to which exactly of the admitted forms

<sup>1</sup> 'Larynxoedem und submucöse Laryngitis,' Berlin, Georg Reimer, 1895.

<sup>2</sup> 'Archiv für pathologische Anatomie, &c.,' vol. cxxxix, Heft 1, 1895, p. 117, *et seq.*

<sup>3</sup> 'St. Thomas's Hospital Reports,' vol. xii, 1882, p. 114.

it belonged in reality, and my attention having thus been rivetted at an early period of my observations of such cases on this question, I have ever since tried to judge the cases which have subsequently come under my observation from an absolutely unprejudiced point of view. The result of this has been an ever-growing conviction *that the various forms of acute septic inflammation of the throat and neck, hitherto considered as so many essentially different diseases, are in reality pathologically identical, that they merely represent degrees varying in virulence of one and the same process, that the question of their primary localisation and subsequent development depends in all probability upon accidental breaches of the protecting surface through which the pathogenic micro-organism which causes the subsequent events finds an entrance, and that it is absolutely impossible to draw at any point a definite line of demarcation between the purely local and the more complicated, or between the œdematous and the suppurative forms.*

Whether this conviction—from the clinical point of view at any rate—is justified or not must be decided from the following short reports of the cases which have come under my observation from 1882 to 1893. If they were arranged in chronological order I feel sure that it would be difficult, if not impossible, to understand how I had arrived at my belief. But if given, as they will be, *in order of ascending severity*, the justifiability of my views will become apparent, I think, to every unbiassed reader. I have only to add that the fact that in most of these cases I saw the patients on one or two occasions only as a consultant must be the excuse for regrettable gaps concerning important points on which some further light is urgently desirable. The few instances in which I had the opportunity of following the cases up more thoroughly are given in full.

CASE 1. *Acute œdematous inflammation of the palate and pharynx; recovery.*—Baron B—, Secretary of the Russian Embassy in Paris, came to me on August 27th, 1882,

complaining of repeated shivering fits, general malaise, great dysphagia, and violent pain in the throat. He had been for a few days under the treatment of Dr. Forsbrook, who had diagnosed œdematous inflammation of the throat and had given him aconite and saline aperients. At the beginning of his illness he was very feverish.

When I saw him he was already better, though there was still very considerable œdematous inflammation of the palate and pharynx, which extended down towards the commencement of the arytaeno-epiglottidean folds, but curiously enough had not affected the latter; the larynx in fact was perfectly normal. No enlargement of the cervical lymphatic glands, no fever, pulse 80, weak. I gave him quinine, and ice internally and externally. The œdematous inflammation disappeared in a few days.

CASE 2. *Acute isolated œdematous inflammation of the epiglottis; recovery.*—I had attended Mr. O. S—, æt. 43, in March, 1882, for slight bronchial catarrh, and had ascertained that his pharynx and larynx were perfectly normal, when two days after his last visit he returned, on March 31st, on account of violent dysphagia, which, without any known cause, had developed within the last two days. The onset of the illness had been attended by slight shivering followed by rise of temperature, and the patient felt very low and ill. On examination enormous isolated œdema of the epiglottis was seen. This part was at least four times as large as it had been before, brilliantly red, smooth, and shiny; the rest of the pharynx and larynx were normal, and there was no enlargement of the cervical glands, no dyspnoea, voice normal. Pulse 106, small, easily compressible. Temperature 100·6°. No cause whatever could be elicited for the attack. I ordered ice internally and externally, quinine, absolute rest, and cold and fluid diet. On the next day there was a distinct diminution of the œdema with relief of the dysphagia, and two days afterwards matters had almost returned to the normal. I have not seen the patient since.

CASE 3. *Acute œdematous inflammation of the posterior wall of the larynx, with slight infiltration of the left side of the neck; recovery.*—Mrs. McS—, æt. 52, was sent to me on June 21st, 1884, by Mr. Johnson Smith, of Greenwich. She had fallen ill about five to six days previously with shivering, febrility, sore throat, and some external swelling of the neck below the left angle of the lower jaw, which shortly afterwards was followed by dysphagia. There had been no difficulty in respiration and only slight hoarseness. She had already commenced to improve before she consulted me. When I saw her, her temperature was normal, pulse 82, very weak, and there was some slight infiltration of the mucous membrane over the arytaenoid cartilages, in the interarytaenoid fold, and on the œsophageal aspect of the cricoid plate. The neck was not tender on pressure, and no tumefaction could be felt on the left side. Under the use of ice externally and internally, and bromide of potassium, recovery took place within a few days.

CASE 4. *Acute œdematous tonsillitis, pharyngitis, and epiglottitis; recovery.*—On March, 3rd, 1888, I was asked by my friend and colleague, Dr. Bristowe, to see with him a relative of his, Mr. S. B—, æt. 26, who had fallen ill five days previously with shivering followed by rise of temperature and sore throat, particularly on the right side. He had been exposed, he spontaneously stated, to sewer gas exhalations. In the first few days of his illness the temperature had been on the average 103°. Then improvement had taken place. Since the 10th, however, the pain in the throat had become aggravated, and he had been almost completely unable to take any food. In the night previous to the consultation difficulty of breathing had set in, followed by great weakness of the voice and very troublesome muco-purulent secretion from the throat, which could only be discharged with much difficulty. On examination the right half of the pharynx, including the tonsil, was seen to be very much swollen,

œdematous, and bluish red. The left half, including the uvula, was less tumefied, but also somewhat swollen and congested. The epiglottis was very œdematous, greatly enlarged, of a brilliant red colour, and quite rigid. Of the interior of the larynx it could only be seen that it was bathed in muco-purulent secretion; no infiltration was visible or appreciable by touch on the outside of the neck. On pressure of the right side of the neck near the larynx there was tenderness, but not acute. The tongue was covered with a thick white fur. The face looked very feverish, and had at the same time an apathetic expression. The temperature was  $101.5^{\circ}$ , the pulse 120, weak, easily compressible. There was no cough. The patient was fully conscious. No affection of heart or lungs.

Dr. Bristowe agreed with me in considering the affection as due to septic influences. Ice internally and externally, quinine, cold fluid nourishment, &c., were ordered.

On the next day the patient was much better. The temperature was  $99.6^{\circ}$ , the pulse 86. He could swallow much more easily. The pharyngeal swelling was much diminished, and had lost its bluish discolouration, and the œdema of the epiglottis had also become considerably diminished. Under continuation of the same treatment the patient got quite well within a few days without any further incident.

CASE 5. *Acute œdematous inflammation of epiglottis and left half of larynx; slight infiltration of left half of neck in a diabetic patient; recovery.*—On December 25th, 1887, I was asked by Dr. Milson to see with him Mr. P—, æt. 47, who had been suffering from diabetes, gout, and albuminuria. There had been a suspicion of renal calculi, and there was a history of syphilis. The patient had had on the 23rd and 24th several shivering fits without any known cause (the drains were stated to be in good order), followed by feverishness, pain in the left side of the neck



close to the larynx, difficulty in swallowing, and since the morning of the 25th also hoarseness. There was no dyspnoea, but an increased amount of albumen in the urine. In the course of the 25th the indisposition increased, and when I saw the patient at 5 p.m. his temperature was  $102^{\circ}$ , pulse 116, hard and resistant; heart and lungs normal. There was some infiltration in the neighbourhood of the left angle of the jaw, which was tender on pressure. The pharynx was normal, the epiglottis much congested, and together with the left ventricular band and arytaenoid cartilage greatly swollen and slightly cedematous. The movements of the vocal cords were normal. Ice internally and externally and quinine were ordered.

On the 27th, when I saw the patient again, I heard that under this treatment improvement had taken place in the two previous days. On the 27th the temperature was normal, pulse 84. The external infiltration had nearly disappeared, and the laryngeal swelling was much diminished. On the 28th, however, again sudden pain in the throat with difficulty of swallowing was experienced, which lasted about an hour and a half, and then disappeared. When I saw the patient at 5.30 in the afternoon nothing abnormal could be ascertained in the throat or externally, but the temperature was  $101^{\circ}$ , and the pulse again 96. The next day everything was normal again, and the patient made a good recovery.

A few days later I received a letter from the patient, in which he informed me in confirmation of the correctness of my suspicion (I had expressed a strong belief that he had been exposed to septic influences) that an examination of the house in which his new offices were situated had disclosed the fact that the drain under it was an old brick barrel-drain, containing a considerable deposit of sewage in direct communication with the sewer, and with openings and rat-holes into the basement story, from which an encased rain-water pipe ran up through his room, the casing forming a duct for the air in addition to

a soil-pipe to a w.-c. in the next room. He further stated that he had only taken possession a few days before the onset of the throat attack. It may be observed here that the patient was an eminent architect himself.

**CASE 6.** *Left-sided tonsillitis; left-sided œdematous inflammation of the pharynx; inflammatory œdema of the epiglottis and of the whole left half of larynx; brawny infiltration of left half of neck; recovery.*—On December 2nd, 1887, I was requested by Mr. Argles to see with him Mrs. W—, æt. 30, who had always enjoyed the best of health, but had fallen ill about a week previously with sore throat. According to Dr. Argles' statements the onset of the disease had been of the nature of undoubted tonsillitis on the left side. For nearly a week apparent improvements and aggravations had alternated. On the evening of the day previous to the consultation, however, the pain, so far complained of as being situated in the neighbourhood of the left tonsil, and the dysphagia, which was localised in the same region, had suddenly travelled further down to the level of the larynx, and a brawny infiltration of the whole left half of the neck from the angle of the jaw to the clavicle had occurred. There had been no rigor or shivering fit so far as could be ascertained, but the patient had been feverish throughout, and the temperature on the morning of December 2nd was 100°, the pulse 120, soft and easily compressible. The drains of the house were stated to be in the best of order. Inquiries as to whether the patient had met with any external or internal injury shortly before the onset of the illness had a negative result.

On examination the whole of the left half of the pharynx was found to be greatly swollen and congested, but nowhere ulcerated; the intensity of the inflammation increased in a downward direction. The epiglottis was enormously swollen, œdematous, brilliantly red, and shiny. The interior of the larynx could on this account not be seen. The voice was slightly hoarse, but there was no

dyspnœa. The dysphagia was very considerable ; the infiltrated portions of the neck were, on the whole, little tender on pressure, mostly so close to the larynx. Lungs and heart healthy ; no evidence of other complications. Ice was administered internally, and cold applied externally by means of Leiter's tube ; five grains of quinine were given every four hours ; and nourishing diet was ordered.

Under this treatment the external and internal swelling in the course of the next few days somewhat decreased, and no metastasis of any kind occurred. The temperature began to fall ; it was on the evening of the 2nd,  $100^{\circ}$ , the pulse 104. In the morning of the 3rd temperature  $98.4^{\circ}$ , in the evening  $101^{\circ}$ , the pulse being 92.

On the morning of the 4th there was less difficulty in swallowing, and no dyspnœa ; slight cough, temperature  $99^{\circ}$ , pulse 102. The infiltration extended down to half the length of the trachea, and was therefore less extensive than on the 2nd ; its hardness also was less. The epiglottis was still much swollen, but not to the same extent as on the 2nd, nor was it so red. Temperature in the evening  $100^{\circ}$ .

On the 5th further improvement had taken place ; temperature in the morning  $98^{\circ}$ , pulse 96. The external infiltration had much diminished ; there was hardly any tenderness on pressure. The patient could swallow better, and the cough which had troubled her during the last day had also diminished. The epiglottis was again less swollen and red. In the evening, however, the temperature again rose to  $101.2^{\circ}$ , and the cough became more troublesome. Some rhonchi were heard all over the chest.

On the morning of the 6th the temperature was  $99^{\circ}$ , the pulse 96. The external infiltration was unchanged ; internally the swelling of the epiglottis had slightly diminished on the right side, but was unchanged on the left. In the evening the temperature was  $98.6^{\circ}$ .

On the evening of the 7th the temperature was  $97.8^{\circ}$ , pulse 92. The patient felt well. The external infiltra-

tion had diminished. The pharynx looked quite normal. The tumefaction of the epiglottis had further diminished also on the left side, and it was now possible to look into the larynx itself and to see that the whole left half was very much swollen. There was much less dysphagia; the cough was still troublesome, but seemed less so than on the previous day. The patient slept well. An iodide of potassium ointment was ordered to be rubbed into the neck.

On the 8th very considerable improvement had taken place. The temperature had become quite normal, the pulse 84 and of good quality. The external infiltration had very considerably decreased. There were no pains, no cough, no dysphagia, and good sleep. The epiglottis was again less swollen; its left half, however, and the left arytaenoid cartilage, were still very oedematous, though somewhat less than on the previous day.

Further improvement had taken place on the 10th. The temperature had remained normal since the 8th, the pulse 80 and of good quality. Externally there was only a small remnant left of the infiltration, corresponding to the outline of the thyroid cartilage. Internally the epiglottis had now entirely returned to its normal dimension, the mucous membrane over the left arytaenoid cartilage was still much infiltrated, but quite pale. The patient got up on that day.

On the 15th the external swelling had entirely disappeared. The left arytaenoid cartilage was still somewhat oedematous. On this day it was ascertained that through a defect in the drainage the sewage from the stables situated behind the house was directly evacuated into the house itself.

On the 20th, when I saw the patient for the last time, the last remnant of oedema could still be seen on the left arytaenoid cartilage, but in all other respects the patient was perfectly well. The urine had been examined throughout her illness, but neither albumen nor sugar had ever been found.

CASE 7. *Acute œdematous inflammation of posterior wall of the larynx; double patchy pneumonia; slight pericarditis; recovery.*—On April 29th, 1892, I was hurriedly sent for by Dr. Arthur Farr to see Mrs. J—, a lady aged about 70, who, after going to bed apparently well on the previous night, awoke in the morning at 6 o'clock with great dyspnœa, which further increased so much that about 11.30 I was telegraphed for to come and be prepared for tracheotomy. On arrival I found the patient propped up in bed, breathing with some but not excessive difficulty, very little stridor, and the voice quite normal. I was informed that she had had a similar attack, though not so bad, some years ago, and that the dyspnœa this time had been much worse, but had already decreased during the last two hours. On examination I found the pharynx perfectly normal, the vocal cords much congested, their posterior ends being close to one another, leaving only a small, elliptic slit for the passage of air, and the posterior wall of the larynx being very œdematous. Rapid up-and-down movements of larynx, no external swelling, no tenderness on pressure, no dysphagia. Pulse 130, very weak and easily compressible. Dr. Farr, who meanwhile had arrived, agreed with me that for the moment tracheotomy was unnecessary. Quinine and ice internally and application of Leiter's bag externally were ordered.

On a further consultation at 3 p.m. of the same day, the aspect of the pharynx and the laryngeal conditions were very much the same. There was no increase of dyspnœa, the pulse was still 130 and very weak and small, whilst the temperature, which had been taken three times since the morning, had always been found normal or slightly subnormal. On the right side of the sternum in the third intercostal space there was a small patch over which distinct fine crepitant râles could be heard, and slight pericardial friction was noticed over the apex. I expressed my conviction that the case was one of septic inflammation, and that the prognosis was serious.

June 30th.—The patient has passed a good night, and her breathing to-day is distinctly easier. The larynx is still congested, but there is much less œdema. The inflammation of the right lung had much extended, and at the time of examination involved the whole upper part of the right upper lobe, whilst also over the base of the left lung tubular breathing was noticeable. There was, however, no pleurisy and not much discomfort in breathing, the number of respirations being only 32. The temperature was gradually rising, and at the time of consultation was a little over  $100^{\circ}$ , pulse about 120. The pericardial friction at the apex was only very faintly audible, and there was no extension of it. There was no albumen and no sugar in the urine, but an abundance of phosphates.

On the following day I had a letter from Dr. Farr to say that the patient was rather better, that the physical signs in the chest had improved, that the respirations were 30, the pulse 110, of fair quality, and the temperature  $100^{\circ}$ . The throat felt comfortable. I afterwards heard from Dr. Farr that the patient had made an uninterrupted recovery.

CASE 8. *Acute œdematous inflammation of epiglottis and arytenoid cartilages (? purulent infiltration); double patchy pneumonia; right-sided pleurisy; recovery.*—On May 5th, 1892, I was hurriedly sent for by Dr. Henry C. Martin to see with him Miss L—, æt.  $14\frac{1}{2}$ , and on arrival heard from him the following history. The child, who had been staying with her parents in the country, had had a fortnight ago a sore throat with high temperature, which at that time appeared to be perfectly epidemic in the family. She had, however, entirely recovered from this, and had come to town on the previous day in order to go to a juvenile party. Whilst there she felt rather indisposed. On her return she had an attack of shivering in the carriage, and felt very cold afterwards. Next morning there was great difficulty and pain in swallowing, hoarse-

ness, gradual loss of voice, increasing dyspnœa. Temperature at 11, 104°, and at 12, 105°. Then Dr. Martin sent for me. On arrival I found her in bed, with very slight stridor, quick respiration (36), pulse between 120 and 130, regular and strong. There was absolute aphonia, considerable pain in throat, tenderness on pressure over larynx, but no brawny infiltration. Pharynx normal; epiglottis enormously swollen and œdematous, bright red. Arytæ-noid cartilages similarly swollen. No view of vocal cords obtained, but enough was seen to show that there was still room enough in the glottis not to necessitate immediate tracheotomy. The organs of chest and abdomen all healthy. I expressed at once the opinion that this was a septic inflammation, and prepared for further complications. A Leiter's bag of ice-water was ordered round the neck, six grains of quinine given every four hours, and twenty grains of bromide of potassium every four hours. At 6.45 o'clock the same evening, when we met again, the temperature had gradually gone down to 102°, the larynx was not so much swollen, but very tender outside, especially on the left side; in the third intercostal space on the right side, close to the sternum, a very limited patch was discovered where somewhat prolonged harsh expiration could be heard. Otherwise the chest was normal.

On May 5th at 8 a.m. I heard, on meeting Dr. Martin, that the patient had had on the whole a comfortable night, and had slept at intervals for some length of time. The breathing was decidedly better. She had been repeatedly sick after taking the quinine, which had consequently only been given in half doses during the night. The tongue was very furred, the voice a little stronger than before, the dyspnœa decidedly relieved. Respirations were about 22, temp. 101°, the urine contained neither albumen nor sugar, the bowels had not acted. There was no pain in the chest. On examination the larynx was seen to be less swollen than on the previous day, but the breath was fœtid, and the neighbourhood of

the arytaenoid cartilages bathed in thin pus. On examination of the chest, the bronchial expiration at the place in front noted on the previous day was more pronounced. Additionally, there was slight dulness over the left apex, and considerable dulness over both bases with very deficient entry of air.

At 11 a.m. a violent rigor occurred, followed by rise of temperature to  $104^{\circ}$ . The patient complained of severe pain in the pit of the stomach. Dr. Martin ordered two grains of calomel and a turpentine-and-gruel enema. Temperature in the evening rose to  $105^{\circ}$ .

At 6.45 p.m. a consultation took place between Sir William Broadbent, Dr. Martin, and myself. The swelling of the larynx had again considerably decreased, and the tenderness outside had also decidedly diminished. Respirations were again 36, pulse 124, and the patient was decidedly weaker, complaining of pain across the chest. In front, over the spot mentioned before, distinct harsh expiration, and in the second intercostal space on the right side a limited patch of pleuritic friction, were noticed. Posteriorly, the inspiratory sounds diminished in intensity below the angles of the scapulæ. On the right side downwards from the scapula there was distinct tubular breathing and crepitation, whilst on the left side a limited patch of the same character just below the angle of the scapula was observed. We all agreed that patchy pneumonia had developed and that the prognosis was serious. It was decided to go on with the quinine, if necessary by the rectum, and to let the patient inhale every three hours some of Brin's oxygen. Stimulants also (iced champagne) were to be taken when necessary. The bowels had acted twice.

May 6th, 8 a.m.—The temperature had gradually fallen through the night to  $99^{\circ}$ . Pulse 96, weak but regular. The patient had had a tolerable night, but complained of pain in front of chest and at the bases of both lungs. She felt rather weak, but had taken milk, medicine, and stimulants freely. On examination the



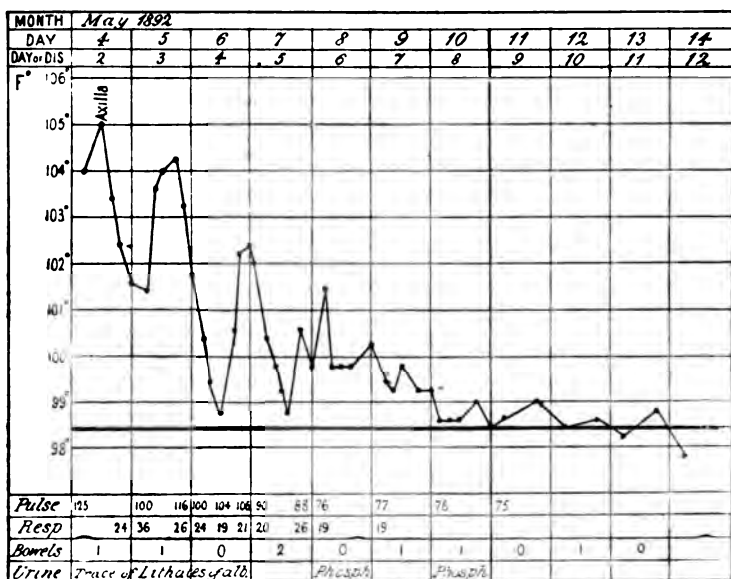
larynx was seen so much better that the ice-bag could be discontinued. The voice, which already on the previous evening had come back to some extent, was only slightly hoarse, but there was still some slight soreness of the throat, which on the previous day had been successfully combated by sucking cocaine lozenges (one-tenth of a grain in each lozenge). The larynx externally was still, but only very slightly, tender on pressure. On examination of the chest there was distinct consolidation over both bases posteriorly from the angles of the scapulæ downwards, more so on right side. In front friction and occasional crepitation could be heard in the third intercostal space on the right side near the sternum, and also over the heart in a very limited area close to the left of the sternum. The general aspect of matters, however, was distinctly better. We decided to continue with the quinine and the stimulants, but to discontinue the ice-bag and to use, if necessary, hot poultices every three hours.

6.45 p.m.—The temperature has again risen to 102°, and the pulse to 114 in the course of the day, but without any further rigor. The patient on the whole feels comfortable, but complains of occasional pains across the chest. The voice is almost normal, the slight soreness of throat still present, the epiglottis still somewhat swollen, especially on the left side, the arytaenoid cartilages much less swollen. Vocal cords normal, their movements unimpeded. Pressure on larynx is still somewhat painfully felt, but distinctly less than before. The physical signs in the chest are very much as they were in the morning. The patient begins to suffer from the effects of quinine, and every second dose is reduced to one half.

10th.—In the course of the last four days nothing remarkable has happened, except that on Saturday morning there was a trace of albumen in the urine, which, however, soon disappeared, and has been replaced by a large quantity of phosphates. The temperature has gone

up in the afternoons (see the following Chart), but has nevertheless been steadily decreasing on the whole. The

CHART I.



congestion of the lungs clears up remarkably quickly. On Sunday morning there seemed to be some slight effusion in the right side, but this also is fast disappearing under the use of iodide of potassium and quinine. The larynx is practically normal, there is no pain, and the child feels in every respect much better.

11th.—During the last two days there has been again steady improvement, the temperature gradually falling until it reached the normal to-day, and the congestion of the bases clearing up. At present the right base is almost normal, and only the inspiratory murmur at the left base is still somewhat weak. The throat is perfectly normal. There has been a slight pain in the left shoulder-joint yesterday, but this has entirely disappeared this

morning. On pressure of the left side the larynx is still very slightly tender. Ordered Ung. Iod. one part to Ung. Pot. Iod. three parts, to rub in externally; and as soon as the temperature has become quite normal, to take phosphate of iron, arsenic, strychnia, and acid glycerine of pepsin.

This was the last I saw of the patient, and I afterwards heard from Dr. Martin that she had made a full and complete recovery.

*CASE 9. Acute inflammatory œdema of epiglottis and arytxno-epiglottic folds following transitory inflammation of pharynx; epileptiform convulsions; ataxic respiration; brawny induration of neck; albumen and sugar in urine; delirium; death.*—My next case has a special and very melancholy interest for this Society, inasmuch as its subject was one of its own Fellows, my late colleague, Mr. Francis Mason, F.R.C.S. In describing his case I shall follow in part the report given in the 'Lancet' of June 12th, 1886, by Drs. Ord and Buzzard, in part my own notes taken at the time.

Mr. Mason, who at the time of his demise was only 48 years old, and who had, with the exception of frequent attacks of tonsillitis, enjoyed remarkably good health, had attended on the afternoon of June 1st, 1886, a Staff Meeting at St. Thomas's Hospital at which I happened to be his neighbour. He told me on that occasion that he had never felt better in his life. The same evening, however, he complained of sore throat. The night was tolerably good, but next morning he did not feel very well and sent for Dr. Ord, who examined him and discovered some swelling and redness in the fauces just below the right tonsil, and, in the belief that probably a fresh attack of tonsillitis of a gouty character was impending, prescribed salicylate of sodium and bromide of potassium for him. Although no improvement took place, the patient did his work till 5 o'clock in the afternoon. At that time, however, he felt so ill that he returned

home and went to bed. Previous to this it is remarkable that he ordered his tracheotomy instruments to be placed in his bedroom in readiness. No rigor occurred at the outset of the illness. Almost immediately, however, after he had gone to bed an attack of dyspnœa of such violence followed that the patient thought he would be suffocated. Dr. Ord, who was immediately sent for, found considerable swelling of the epiglottis and sent for me. I arrived at 7.30 p.m. and found the patient sitting upright in bed, the face covered with cold perspiration, but not cyanotic. He was absolutely voiceless, and complained of great dysphagia, so that drinking, even of small quantities of water, caused violent pain in the throat, particularly on the right side at the level of the larynx; he also suffered from a peculiar dyspnœa, or, more correctly said, "hunger for air." There was at that time not the least laryngeal or tracheal stridor. The temperature was  $103.5^{\circ}$ , the pulse small, most rapid, *i. e.* 140 to 150 beats in the minute, and very compressible. The neck was tender on pressure on the right side near the larynx. External swelling, however, was hardly to be noticed, and only in considerable depth a certain hardness was felt to the extent of about one inch on the right side of the trachea. In the pharynx at that time absolutely nothing abnormal could be seen, neither swelling nor discolouration. The epiglottis, on the other hand, was changed into an enormous, vividly red-coloured, œdematous tumour, which did not permit of the interior of the larynx being inspected.

I at once told Dr. Ord that I was convinced this was a case of erysipelas of the larynx, and Dr. Ord agreed with this opinion. I energetically scarified the epiglottis with a guarded laryngeal knife, and suggested the application of a Leiter's tube with an ice-water current, sucking of small lumps of ice if possible, sulphate of quinine in five-grain doses every four hours, and an aperient. The scarification was followed by expectoration of a large quantity of dark blood, and the patient felt a little easier, but ex-

pressed his firm conviction that his illness would end fatally. The respiration retained its peculiar character. Shortly after the incision the pulse was still 140; the temperature, however, suddenly fell to  $99.5^{\circ}$ .

At 10 o'clock at night the swelling on the right side of the neck was much more marked, and there was some dusky discolouration of the skin corresponding to it. Watch was set, in which the assistant surgeons of St. Thomas's Hospital alternated. For some hours after the scarification the patient remained easier, but about 2.30 a.m. on the following morning he suddenly ceased to breathe. Mr. Pitts, who was watching him, at once performed tracheotomy under circumstances of great difficulty, only Mr. Mason's wife being present at the time. After the tube had been inserted there followed an attack of epileptiform convulsions. These presently passed off, and the patient recovered consciousness and was evidently greatly relieved, but the pulse remained very quick, ranging above 160, and the respiration was irregular, and, so to speak, ataxic, resembling, but not quite corresponding to, Cheyne-Stokes respiration. On the next morning the brawny induration of the neck extended already to both sides, and up to the death of the patient still further increased, without showing anywhere distinct signs of suppuration in the depths of the tissues. The dysphagia on Friday morning was as great as before. The epiglottis had slightly shrunk at that time, and the two spots where it was scarified the evening previously were distinctly visible. It could now also be seen that the arytaeno-epiglottidean folds were enormously swollen. The interior of the larynx proper, however, was still invisible. After this it was impossible to make any further laryngoscopic examination. During the morning of this day, Friday the 4th, the pulse fell in quickness and rose in strength, but the breathing never became quite natural in rhythm. In the evening the improvement ceased and delirium set in. Early on Saturday morning Mr. Mason again appeared to revive, and to a casual observer a remarkable improvement might

have seemed to have taken place: his voice was perfectly clear, the dysphagia had entirely disappeared (our poor friend drank in the presence of all his colleagues assembled for consultation a glass of champagne, in order to show us that he could drink without any difficulty), and pressure on the neck was, in spite of the existing enormous swelling, quite painless, but even at that time his mind was evidently not clear, and the irregularity of the breathing and of the heart's action continued. The revival was transient, and he died at 1.50 on Saturday afternoon, the illness having thus from the very first symptom to the fatal issue occupied not more than sixty to seventy hours. The urine examined in the night of Thursday was loaded with albumen and also contained much sugar; chlorides were suppressed. Under the microscope there were seen granular and hyaline casts, and later on some blood-corpuscles. It is possible that there may have been some interstitial change in the kidneys, but probably the greater part of the albumen and of the sugar were present only as the result of the intensely acute septic attack. On Saturday morning the albumen had diminished very greatly, and the sugar reaction could hardly be obtained. There was at no time any evidence of disease of the lungs or of any of the serous membranes. No post-mortem examination took place.

CASE 10. *Acute œdematous inflammation of (?) pharynx, epiglottis, arytxeno-epiglottidean folds, and mucous membrane over arytxenoid cartilages; left-sided basic pneumonia; death.*—On May 31st, 1890, I was asked by Dr. M. H. Neale to see with him Mrs. A—, æt. 60, who on the previous evening had had a rigor, followed by great pain in the throat, complete aphagia, hoarseness, great dyspnœa, collapse, diarrhœa, and slight delirium. All this had, at the time when I saw the patient, slightly improved. There was no record of temperature, nor had the urine been examined for albumen and sugar. From the history of the attack it deserves to be mentioned that

the patient on the day previous to the occurrence of the rigor had kissed a child, which had been dead for several days, in its coffin.

On examination at 5.30 p.m. there was orthopnoea but no laryngeal dyspnoea, cyanosis of the lips, yellow sodden appearance of the face, moaning respiration, hoarseness but no aphonia, great dysphagia, mental powers slightly obscured. The temperature was  $100.8^{\circ}$ ; the pulse about 120, extremely irregular. There had been no repetition of the initial rigor.

Over the thorax everywhere rhonchi were heard, but there was no evidence of pneumonia, pleurisy, or pericarditis. The heart-sounds were dull and irregular. The mucous membrane of the pharynx appeared much wrinkled, but not swollen. The epiglottis, the arytaeno-epiglottidean folds, and the mucous membrane over the arytaenoid cartilages were enormously swollen, of a dull red, in part covered with tenacious muco-purulent secretion; the vocal cords normal; not much stenosis of the air-passages. Great tenderness on pressure on both sides of the neck, but nowhere any infiltration.

I gave a very serious prognosis, and advised the external and internal application of ice, quinine, nourishing fluid diet after previous cocainisation by means of a 20 per cent. spray, and, if necessary, tracheotomy.

On June 3rd I received the following letter from Dr. Neale:

"Mrs. A—, the patient you saw with me on Saturday afternoon, died last evening. After you saw her the pulse improved very much; it became regular for some hours. Then on Sunday the respirations increased, and there was marked tubular breathing at the base of the left lung. The temperature did not rise above  $100.7^{\circ}$ . The throat symptoms improved slightly, and once or twice on Sunday she swallowed without much trouble. The cocaine, however, did not act well, though I painted and sprayed her well with 15 per cent. solution. I am very sorry I could not examine the urine. Very little

was passed after you left, and always some fæces with it. She was too weak to use a catheter."

CASE 11. *Acute œdematous inflammation of tonsils, pharynx, and larynx; double basic pneumonia; death.*—This case is the most terrible of all I have seen, with regard to the rapidity of its course and its peculiar circumstances, and will justify, I think, my entering a little more fully upon its details.

On June 5th, 1888, a consultation had been arranged between Mr. T. S. Townsend and myself to take place at his house in the afternoon, in order to examine a lady who suffered from some affection of the singing voice. Knowing the nature of the case beforehand, I only took a laryngoscope and mirror with me. When I arrived at Mr. Townsend's house I found the patient only, and a hurried note from himself to the effect that he dared not leave a patient, Sir P. M—, who was suffering from acute laryngitis, and who, he feared, would require tracheotomy to be performed. He added that he wished me to come to Sir P. M—'s residence, which was near by, as soon as I had finished my examination of the lady's throat.

In compliance with his request I hurried to the patient's residence, and found him a strong, fine man of about 56, suffering from orthopnoea, bathed in perspiration, the face dusky, all superficial veins standing out. With him were Mr. Townsend and Mr. Arthur Durham, who had also been sent for. On inquiry I learned that the patient on the previous day had gone in the best of health to Eton to celebrate the 4th of June with his son, who was at school there. He had returned perfectly well, but had awoke early in the morning of the 5th with a violent rigor, followed by rise of temperature, which when taken early in the morning had already been 104°. Shortly after rapidly increasing difficulty of swallowing, which soon amounted to complete aphagia, had made its appearance. He had begun to perspire strongly, and had continued to do so ever since.



About 10 o'clock in the morning Mr. Townsend, who had been sent for, had found tonsillitis and œdema of the larynx on the left side and commencing pneumonia in both bases. By that time the patient could not even drink water. The hoarseness also rapidly increased, and soon amounted to complete aphonia. This in turn was quickly followed by difficulty in breathing, and at 3 o'clock there had been a severe attack of choking. The temperature at that time was 103°. When Mr. Durham, Mr. Townsend, and I examined the patient at 4.30 p.m., there was no longer any swelling of the left tonsil to be seen, but bluish discolouration of the whole pharynx. The larynx was also bluishly discoloured so far as visible (epiglottis and arytaenoid cartilages), enormously œdematous, and covered by frothy secretion. The patient constantly hawked and expectorated partly rusty, partly hæmorrhagic sputa. The neck externally was tender on pressure only over the region of the larynx itself, but no infiltration could anywhere be felt. The heart-sounds were dull but regular; the pulse 80, regular. The urine had not been examined. An examination of the bases of the lungs could not then be made on account of the serious condition of the patient. The dyspnoea at 4.30 was moderate, there was some stridor, but no extensive respiratory excursions of the larynx.

We all three agreed as to the septic character of the disease, but considered tracheotomy not immediately required, particularly in consideration of the serious prognosis anyhow. We advised ice internally, warm poultices round the neck (by Mr. Durham's wish), and nutritive enemata with quinine, as the patient could not swallow anything. Mr. Durham then left the house, and I was about to do the same, when Mr. Townsend requested me to explain to Lady M— (who he knew was much opposed to tracheotomy under any circumstances) how matters stood, and to ask her not to put any difficulties in his way, should the operation after all become unavoidable. I of course complied with his request, and spoke to Lady M— in an

adjoining room. During our conversation the patient had a sudden attack of asphyxia, and both pulse and respiration stopped. I was called in immediately, and, whilst the patient was moribund, performed tracheotomy with my penknife, no other instrument being at hand. I succeeded in rapidly opening the trachea, and held the edges of the wound asunder with bent hairpins, which the patient's daughters, who, as well as his wife, were in the room, supplied, Mr. Townsend meanwhile carrying out for fully fifteen minutes artificial respiration; but all was in vain, the patient was dead. The whole course of the disease, thus, from the initial rigor to the fatal end occupied no more than eleven hours. No post-mortem examination was made.

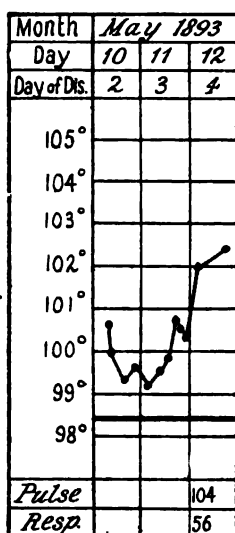
CASE 12. *Brawny infiltration of neck; congestion of pharynx; general inflammatory œdema of larynx; left-sided pleurisy and pneumonia; death.*—On November 24th, 1885, I saw, with Dr. Chepmell, Mrs. S—, æt. 60, a lady whose only son had just been killed in the Soudanese War, whose general health had greatly suffered since her bereavement, and who had spent all her time in visiting wounded and sick soldiers returned from the war in the Military Hospital. It was known that amongst them there were several septic cases. On November 20th she had a rigor, followed by pain in the throat, shortly afterwards by dysphagia, and finally by dyspnœa. When I saw her in the morning of the 24th there was very great dyspnœa, tracheal stridor, cough, and sero-purulent expectoration; the temperature was  $101.5^{\circ}$ , the pulse 120. There was great prostration, and the patient sweated profusely. Her face was of a dusky colour, the sensorium rather obscured. The entire neck presented brawny infiltration, but without much discolouration. The pharynx and larynx were greatly congested, the latter slightly œdematous all over. The breath-sounds were rough, but otherwise normal; no abnormal dulness anywhere over the thorax. A Leiter's tube was applied round the

neck, and ice was given internally, together with an expectorant; a croup tent was erected over the bed, and plenty of nourishment and stimulants administered. My prognosis was very serious. In the evening of the same day the breathing was slightly better, temperature, however,  $102^{\circ}$ , and pulse 128. The patient was very restless. Quinine (5 grains every four hours) was added to the medication. After a restless night the temperature, taken in the rectum, on the morning of the 25th, was  $103^{\circ}$ ; the pulse 132, very weak, slightly irregular. The patient complained of violent headache and frequent sickness. The breathing and the expectoration were easier, but there was distinct dulness both in front and posteriorly over the base of the left lung, where also crepitation and pleuritic friction were heard. The patient was evidently sinking. In the afternoon a consultation was held with Sir William Gull, who corroborated the bad prognosis I had given in the morning. The temperature then was  $104^{\circ}$ , and the patient very comatose. Death took place the same evening. No post-mortem examination was held.

CASE 13. *Acute œdematous inflammation of epiglottis, aryteno-epiglottic folds and mucous membrane over arytenoid cartilages; abscess over left arytenoid cartilage; brawny infiltration of neck; double pleurisy; delirium; attempted suicide; death.*—On May 11th, 1893, I was requested by my colleague, Dr. Payne, to whom I am much obliged for permission to make use of this case, to see in Arthur (small) Ward of St. Thomas's Hospital, T. B—, æt. 37, a porter at one of Messrs. Spiers and Pond's establishments, who had been admitted on the previous day on account of constriction and soreness of throat of one day's duration, with inability to eat or to drink. He had previously enjoyed good health, and had only once, three years before, suffered from bad sore throat and a submaxillary swelling, which had been incised in Westminster Hospital. His present illness began on May 9th with the symptoms mentioned. About twelve

hours later he had a shivering fit, with aggravation of the symptoms. He complained much of the offensive odours of the refuse which in his capacity as porter he had to carry about.

CHART II.



Temperature chart of Case 13 from admission until death.

On admission, at 10.30 p.m., the patient spoke with a low gruff voice, metallic in quality, coughed constantly, and showed evidence of respiratory obstruction.<sup>1</sup> His pharynx was normal, the epiglottis slightly red and swollen, the arytaenoid cartilages and the arytaeno-epiglottidean folds greatly swollen, deep red, and evidently acutely inflamed. The vocal cords were slightly red, and their abduction impaired. There was much muco-pus in the larynx. Externally a brawny inflammatory oedema extended from the lower jaw to the clavicle on the right

<sup>1</sup> For the notes of this case and of the post-mortem examination I am indebted to Mr. Box, Medical Registrar to St. Thomas's Hospital.

side. At 12.45 a.m., *i. e.* about two hours after admission, tracheotomy became necessary, and was successfully performed in spite of much lividity and of free venous hæmorrhage. At the same time the inflammatory swelling in the neck was incised, but only serum escaped. The temperature on admission had been  $100.6^{\circ}$ , at 4 p.m. it was  $100^{\circ}$ , at 8 p.m.  $99.4^{\circ}$ , at midnight  $99.6^{\circ}$ . During the next twelve hours after tracheotomy the patient expectorated about one ounce of thin, dark coloured, but not offensive pus.

On May 11th at 10.30 a.m. his voice was much improved. In the larynx an abscess over the left arytaenoid cartilage had burst, and the swelling had largely subsided, so that the cords could be seen for their entire length; they moved well. Deglutition was still painful. The tracheotomy tube was removed, and the interior of the larynx painted with Tinct. Ferri Perchloridi. There was still much cervical swelling and tenderness, but no purulent discharge.

In the afternoon I was requested to see the patient, and made the following note: "pharynx free, epiglottis still thickened, the mucous membrane is corrugated and bright red, the swelling of the arytaenoids is hardly noticeable, the movements of the cords are free, much tenacious pus in larynx."

In spite of the apparent improvement and of the absence at that time of any thoracic or other complication, I expressed the belief that the case would take a very serious course. The temperature during the 11th was at 4 a.m.  $99.2^{\circ}$ , at 8 a.m.  $99.6^{\circ}$ , at noon  $99.8^{\circ}$ , at 4 p.m.  $100.8^{\circ}$ , at 8 p.m.  $100.6^{\circ}$ , at midnight  $100.4^{\circ}$ . On the 12th the temperature at 4 a.m. was  $100^{\circ}$ , at 8 a.m.  $101.6^{\circ}$ , at noon  $102.6^{\circ}$ , and at 4 p.m.  $102^{\circ}$ .

The next note is dated the 12th, 5 p.m.: Respiration 56, pulse 104, temp.  $102^{\circ}$ . The patient was then delirious. There was evidence of right pleurisy and (?) pneumonia. The patient attempted suicide by stabbing himself with scissors, and was very violent. At 8 p.m.

the temperature was  $102.4^{\circ}$ . Death occurred somewhat suddenly at 12.15 a.m.

The post-mortem examination, which has an additional melancholy interest in the fact that it was the last which was performed by my late lamented colleague, Dr. Hadden, is summarised as follows :

Recent pericarditis with a little turbid effusion. Heart normal. Recent lymph over both lower lung lobes and corresponding costal pleuræ. Lungs simply congested. Liver cloudy. Spleen large and soft. No pus in tissues of neck. Much thickening of pillars of fauces, epiglottis, and ary-epiglottidean folds. A small grey slough on the posterior extremity of the right arytæno-epiglottidean fold, covering a small abscess cavity and extending in the direction of the arytænoid cartilage. Interior of the larynx red, swollen, and covered with thick mucus. Much mucus in trachea and bronchi. A small slough involving mucous and submucous coat over a tracheal ring just below the tracheotomy wound (? pressure of tube). Uratic deposit in right great toe-joint; no pus in joints. No record of examination of brain.

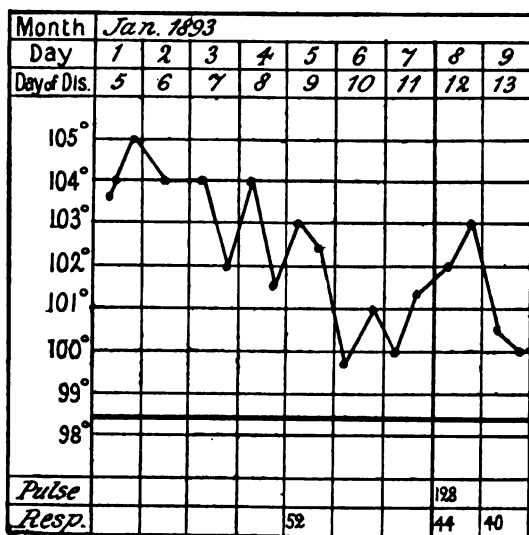
I deeply regret that owing to a mistake I was not informed of the death of the patient nor of the date of the post-mortem examination, and therefore was not present at the latter. The specimens unfortunately were not preserved.

After the patient's death a culture was taken from the secretion of the tracheotomy wound on agar by the house physician, handed to Mr. Shattock, and at once placed in the incubator. A pale yellowish growth developed within the next few days, which on microscopic examination proved to be a *Staphylococcus*. Sub-cultures of this were made in broth, and the fresh cultures injected subcutaneously in the ears and backs of rabbits. No local or general reaction ensued. The experiment was repeated several times with different broth-cultures, but always with negative results. Finally a rabbit was

fed with a large quantity of broth culture, but with equally negative result.

CASE 14. *Brawny induration of neck; follicular tonsillitis; acute inflammatory œdema of epiglottis and left arytxenoid cartilage; deep-seated abscesses in neck; double pleurisy; peritonitis; superficial abscesses; death.*—This is the case from which, as repeatedly mentioned, my occupation with the subject practically dates, and which I have described in vol. xii of the ‘St. Thomas’s Hospital Reports.’

CHART III.



Temperature chart of Case 14 from admission until death.

It is therefore only necessary here briefly to repeat that the patient was a man æt. 31, who came on December 29th, 1882, to St. Thomas's Hospital with a swelling occupying the whole left upper half of his neck, which was tender on pressure, hot, œdematous, and of an erysipelatous redness. At the same time there was acute

follicular tonsillitis and enormous cedema and discolouration of the epiglottis and the left arytaenoid cartilage. The patient was taken into the hospital. He soon became delirious; his temperature varied between  $105^{\circ}$  and  $100^{\circ}$ ; pleurisy developed, and abscesses in the left side of his neck as well as on the hands; and he died on the 9th of January, 1893. An autopsy was made, and the post-mortem diagnosis was: pyæmia ex causa ignota, abscesses in neck, cedema of larynx, double pleurisy, peritonitis, superficial abscesses.

For further details I must refer readers to the original.

The foregoing series of cases represents my personal observations of acute septic inflammations of the throat and neck. If these observations had to be classified according to the nomenclature more or less generally adopted at present, I have no doubt that they would be referred to chapters which are kept entirely distinct from one another in most recent text-books. Case 1 would no doubt go down as an instance of "violent catarrhal inflammation of the palate and pharynx," Case 2 as acute "epiglottitis," Case 3 as "idiopathic cedematous laryngitis," Case 4 as "violent catarrhal inflammation of the tonsils, pharynx, and epiglottis;" although in this case already the likelihood of a septic origin would probably become apparent to many observers. In Case 5 again an experienced observer would no doubt suspect from the fact that one half of the larynx only was in a condition of cedematous infiltration, and that at the same time some slight infiltration of the left half of the neck existed, that the origin of the disease was septic. But differences of opinion might arise as to whether this case had better be called a case of septic laryngitis, of erysipelas of the larynx, or of Ludwig's angina with participation of the larynx. Difficulties of a similar nature present themselves in Case 6; but I think that most observers, if they had seen the patient for the first time at the period of the illness at which she came under my observation,



would not have hesitated to call it a case of erysipelas of the larynx. The next Cases, 7 and 8, would, I believe, be unhesitatingly classified by most members of the profession as instances of erysipelatous inflammation of the larynx with subsequent extension to the thoracic organs, although in Case 8 the fact that at one time the larynx was bathed in thin pus could not but raise the question whether this was a pure erysipelatous inflammation, or whether there was possibly at the same time a purulent infiltration of invisible parts of the upper air-passages. In Case 9, again, to any observer who had not watched the case from the beginning the difficulty of deciding whether it was a case of erysipelas of the larynx with subsequent extension to the cellular tissue of the neck and early and severe involvement of the central nervous system, or *vice versa*, whether it was a case of Ludwig's angina with subsequent extension to the larynx and implication of the highest nerve-centres—would have been very considerable, although in view of the fact that the development of matters fortunately was carefully watched by competent observers from the very onset leaves hardly any doubt that the former of the two alternatives corresponded to the actual facts. Cases 10 and 11, I think, would have been looked upon as instances of erysipelas of the larynx followed by pneumonia. In Case 12 again we meet with the difficulty—this time greater than ever—of deciding whether, according to the present nomenclature, it should be classified as an example of Ludwig's angina or of erysipelas of the larynx. Case 13 would probably be regarded by some as an example of erysipelas of the larynx, by others as one of phlegmonous laryngitis, and again by others as an example of Ludwig's angina; whilst in Case 14, finally, as already stated in a previous paragraph, good reasons could be adduced for classifying it under any of the headings—erysipelas of the larynx, Ludwig's angina, deep-seated phlegmon of the neck.

It will have been seen from this list that my personal observations do not appear to include a typical example

of the affections described by Carrington and Hale White and by Senator under the titles of "phlegmonous pharyngitis" or as "acute infective phlegmon of the pharynx," *i. e.* cases in which a diffuse purulent infiltration in the deeper tissues of the pharyngeal mucous membrane existed, which thence is propagated to the larynx and the glands, and secondarily also invades other organs. Apart, however, from the fact that the clinical appearances and the course of the disease in several of my more severe cases closely resemble the descriptions given by Senator himself of his own cases, it is quite possible that if a post-mortem examination had taken place in all my fatal cases, conditions similar to those described by the authors just named might have been found. My two last cases, at any rate, plainly show that an inflammation of the pharynx and larynx which clinically has all the appearances of erysipelas may be accompanied by formation of pus in the neighbourhood of the oedematous parts (in Case 13 in the larynx itself, in Case 14 between the muscles of the neck), and I think it will be generally conceded that no rational differentiation could be established on the sole strength of the fact that in one instance an apparent infiltration exists in the deeper tissues of the pharynx, and in the other in such close neighbourhood to that part as in my two last cases.

But all the difficulties mentioned in the last paragraph are, I venture to believe, of our own making, and will disappear when we have the courage to disabuse our minds of the artificiality of the present system, and to analyse, untrammelled by dogmatic teaching, the essential facts adduced in my observations from the point of view of an unprejudiced critic.

Now what are these facts?—We have before us a number of cases of acute inflammation of the throat and neck, attacking in most instances previously healthy persons, varying enormously in intensity, varying also in primary localisation and in the fact that in some of them the inflammation remained limited to the part or parts

originally attacked, whilst in others an extension occurred, either by contiguity or by metastasis to other organs, and, finally, varying in the character of the exudation, inasmuch as in most cases, so far as could be ascertained, it was of a serous, and in two cases at any rate of a partly purulent nature, whilst in one case (Case 13) a fibrinous exudation accompanied the pleurisy, which was one of the complications set up by the disease. But all these cases were during life characterised by symptoms which both subjectively and objectively not merely resembled one another, but which in reality formed a practically uninterrupted scale of increasing severity of one and the same process, namely, of a septic inflammation characterised by œdematous infiltration of the tissues attacked. It appears to me not merely the simplest, but also the most natural explanation, and one entirely in concord with the general state of our present knowledge of septic inflammations, that the differences in severity, extension of the process and nature of the exudation (*i. e.* whether serous, fibrinous, or purulent) observed in the different cases, depended upon various degrees of *virulence*, and probably also on the *quantity* of the pathogenic micro-organisms which in the individual instances caused the disease, whilst the accident of *primary localisation* was in all probability in each case determined by accidental breaches of surface forming the suitable portal for the entrance of these micro-organisms into the body. There appears to me not the least necessity for subdividing the cases—and thereby merely confusing the issue—into rigid conventional forms, which do not stand, as my series so plainly shows, the test of practical experience; and so strongly am I convinced of the unity of the different forms, that on the strength of my own observations, joined to Carrington and Hale White's and Senator's experiences, I venture to submit, in the following lines, a sketch of the clinical course of these septic inflammations of the throat and neck, which, though no doubt as yet not quite complete, may serve as a skeleton of the clinical and pathological

events, to be invested hereafter by future observers with more complete details.

I should describe the course of events in these cases as follows :

Acute septic inflammations of the throat and neck may be caused by the action of *various* micro-organisms (on this point more in conclusion of this paper), and vary in primary localisation, extent, and complications in accordance with the virulence and probably also the quantity of the pathogenic factors. They may, and do, attack previously healthy persons of both sexes, and apparently of all ages. Nothing definite is as yet known about the length, and indeed the existence, of an incubation stage, whilst prodromal symptoms, though apparently present in some cases and manifested by general malaise, headache, sore throat, and some febrility, may in other, and indeed in the worst cases, be entirely absent. The onset of the disease is usually very sudden, and is manifested by a rigor followed by quick rise of temperature ; but this rigor, too, may not be present, or may be only of the nature of slight shivering, or may occur after some of the actual symptoms of the affection have already made their appearance. Very rarely repeated rigors occur, and, if so, point to further complications. The course of the fever in these cases does not appear to follow any definite rule, and very probably depends upon the virulence of the septic infection in every individual case. In some cases, though of a severe type, it does not rise at all much over  $100^{\circ}$ , whilst in others it goes up to  $105^{\circ}$  and more. The only general conclusion so far admissible seems to point to the fact that the highest temperature in most cases is reached at the very onset of the disease, and that thereafter it gradually falls, although in the event of further complications taking place a renewed rise of temperature may accompany their onset. The pulse very rarely reaches excessive frequency, and during the acute stage of the disease as a rule varies between 110 and 130. It is characterised in most cases

by weakness and by being easily compressible. An irregularity of the pulse and of respiration appears to occur in very rare cases in which an early involvement of the central nervous system has taken place.

With regard to the subjective complaints, the first and almost general one is that of pain in the throat and great difficulty in swallowing, which within a few hours usually amounts to complete aphagia. This difficulty appears to be always present, whether the original seat of the septic inflammation be in the pharynx, the larynx, or the cellular tissue of the neck. It is, in those cases in which the larynx is involved or becomes so in the further progress of the inflammation, followed by hoarseness, loss of voice, and dyspnoea, which may or may not bear a laryngeal character and be accompanied by stridor. These symptoms are attended by a general feeling of great malaise and prostration. The aphagia lasts in some cases for hours, in others for a day or two, but in all cases in which recovery takes place is distinguished by remarkably quick subsidence, as are, indeed, all the other subjective symptoms complained of.

The objective symptoms vary, of course, in accordance with (a) the localisation and (b) the extension of the inflammatory process. In the great majority of cases it appears that the pharynx and particularly the tonsils are the parts first attacked, forming as they do the natural portal for the entrance of micro-organisms into the body. (This point will be more fully discussed further on.) Needless to say, however, it is quite possible that the pathogenic organisms may pass these parts and primarily settle in any other locality in the throat in which an accidental breach of surface favours their entry. Thus, in some cases the larynx—and here above all the epiglottis,—and in a few others the cellular tissue of the neck, will be the original seat of the septic inflammation.

In those cases in which the tonsils and pharynx are first affected these parts very quickly swell to a considerable degree. The inflammation of the tonsils does not

seem to be distinct in anything from ordinary follicular tonsillitis; if the posterior wall of the pharynx, however, be affected, the swelling is much more developed and of a much more œdematous character than in ordinary catarrhal inflammation, and the colour of the inflamed part, whilst in the majority of cases of a bright scarlet, in some instances shows a distinct bluish, sometimes almost dark blue hue. The swelling may remain limited to the pharynx, and after a day or two retrogressive changes, and finally recovery, may take place without the larynx ever becoming affected. If the swelling has been very considerable, the mucous membrane of the affected portion during the retrogressive changes shows a wrinkled appearance. In the very worst cases in which the pharynx has been the primary seat of the disease (cases such as have been described by Carrington and Hale White and by Senator), the whole brunt of the inflammation appears to fall upon that part, and in such cases (in which generally the disease ends in death), after a few days a diffuse purulent inflammation is found in the deeper tissues of the pharyngeal mucous membrane, the inflammation being propagated to the larynx and the glands and secondarily also invading other organs. Needless to say the septic inflammation may spread from the pharynx in other directions than downwards, and may for instance attack the nose, the naso-pharyngeal cavity, the external integument, or the brain. A case in which a phlegmonous pharyngitis was followed by purulent meningitis has recently been reported by von Stein.<sup>1</sup>

In the great majority of cases, however, it is characteristic of the inflammation, when beginning in the pharynx, that it appears to completely leave this part after a few days or even hours and to extend further downwards to the larynx. The previous observations so far recorded all point in the direction that if the latter organ be attacked by septic inflammation, be it by propagation from the pharynx or be it primarily, the epiglottis

<sup>1</sup> 'Monatschrift für Ohrenheilkunde, &c.,' No. 10, 1894.

appears to be singled out prominently to bear the brunt. Next to this part of the larynx the arytaeno-epiglottidean folds and the mucous membrane over the arytaenoid cartilages appear to suffer, whilst not much can be stated as yet with regard to the conditions of the interior of the larynx, the swelling of the vestibule, as a rule, preventing inspection of the vocal cords. From the hoarseness and aphonia, however, attending in many instances the laryngeal inflammation, it is very likely that the interior of the larynx, though possibly to a lesser degree, is also involved in the inflammatory process. The appearance of the epiglottis, arytaeno-epiglottidean folds, and arytaenoid cartilages in these cases is characterised by enormous tumefaction, in consequence of which particularly the first-named part is enlarged to four times or even more its natural size, and presents itself as a semi-transparent scarlet or bluish-red roll, lying across the pharynx. Such dimensions does this swelling sometimes assume, that even the arytaeno-epiglottidean folds and the arytaenoid cartilages are quite concealed from view.

At this stage, in the great majority of cases, there is steadily increasing dyspnoea, aggravated not rarely by attacks of suffocation, which, if not in time relieved by tracheotomy, may end fatally. In very severe cases the exudation becomes partly purulent, and either a diffuse purulent infiltration of some parts of the larynx may be met with, or a distinct abscess may form, which may burst spontaneously, as in Case 13 of my list. In a number of cases the inflammation remains limited to the larynx; in other cases, however, it extends from this part to the neighbouring tissues of the neck, and causes an infiltration of these parts, which may concern either the immediate neighbourhood of the larynx only, or extend over the whole of one or even both sides of the neck. The skin of the neck in such cases is hot, and so tense that it is difficult to make out any details within the general infiltration. The parts are also, as a rule, very tender on pressure. The infiltration is hard, brawny, and its colour varies from slight redness to a

dusky violet. In the worst cases it extends from the lower jaw to the clavicles or even further down, and the outlines of the neck may be completely lost. An erysipelatous redness is found in such cases to extend even further than the infiltration itself. The exudation into the cellular tissue of the neck in most cases of this kind seems to be of a purely serous character. In other cases, however, either diffuse purulent infiltration is met with, or localised abscesses exist in the cedematous cellular tissue or between the muscles of the neck. In the very worst cases metastatic abscesses occur either in superficial parts or in joints.

Whilst the purulent variety of the septic inflammation usually leads to speedy death, cases of serous inflammation of the larynx and its neighbourhood may get well, however considerable the swelling has been, within a few days. Here, again, it is characteristic that the maximum of the inflammation usually is attained within a few hours from its very onset; and that in the cases in which recovery takes place, already in a day or two after the commencement considerable diminution of the swelling forms the rule. Within a few days nothing but a wrinkled appearance of the mucous membrane may give evidence of the enormous swelling which only shortly before formed the most conspicuous sign of the severe illness.

Often, however, the disease does not remain limited to the neck, but spreads—sometimes with incredible rapidity—to other parts. In addition to the lungs, the serous membranes particularly are liable to become attacked, and pleurisy, unilateral or double, pericarditis, peritonitis, and as already mentioned, meningitis, may be developed within a few days or even hours from the initial rigor. When the lungs are attacked, either basic or patchy pneumonia in one or both lungs shows their involvement in the pathological process. Not rarely several of these complications are met with in one and the same case. The exudation in the serous membranes may be either of a serous, fibrinous, or purulent character; hæmorrhagic effusions have not been met with in



my own observations. Even in cases complicated by pneumonia, pericarditis, and pleurisy, recovery is possible, and, if occurring, is remarkable for its quickness and completeness. In more severe cases, however, death ensues under signs of increasing coma and heart failure, and in the worse cases of this character the whole process from its beginning to the fatal end may not occupy more than ten to twelve hours.

In very rare cases it appears as if the whole brunt of the septic infection, apart from the parts first attacked, fell upon the central nervous system. In such cases epileptiform convulsions, delirium, irregularity of heart and pulse action, are amongst the earlier symptoms, and death may occur under signs of severe septic infection of the nervous system, without any complications developing in the chest, and after the local inflammation of the pharynx and larynx has already completely subsided.

In a final series of cases—those which have been hitherto described as cases of angina Ludovici—the primary localisation of the inflammation is the cellular tissue of the neck. The pathogenic micro-organism may penetrate into this part either through an external breach of surface or through a carious tooth, through the tonsils, the lymphatic glands of the tongue and pharynx, &c., &c. In these cases the brawny infiltration of the neck will be the first objective symptom observed, and the participation of the pharynx and larynx only occur at a somewhat later period. In other respects, however, the events observed are absolutely identical with those seen in the cases in which the pharynx or larynx were the parts first attacked.

The foregoing description may serve as a clinical sketch of our present knowledge of these septic inflammations of the throat and neck. It shows, I hope, that my conception of the unity of these various processes is not an arbitrary one, but one fully in accordance with the clinical and pathological facts which have come within my own knowledge.

Whilst thus convinced of the pathological identity of these various processes, I am, needless to say, fully aware that a number of important objections could be raised against my views, and I am myself conscious of the following four which demand an immediate reply :

1. That I had given no proof that the milder cases in my list were really of septic origin, and that the symptoms observed in them may well have been the result of a simple catarrhal inflammation.

2. That the different localisation in these cases, namely, whether originating in the pharynx, larynx, or cellular tissue of the neck, spoke against their being identical.

3. That the variations in the fever curve in the individual cases also seemed to point in the direction that this fever was caused by different, not by identical, processes.

4. And above all, that the fact of the exudation sometimes being of a serous and sometimes of a purulent character most powerfully combated the view that these inflammations were identical in nature.

1. With regard to the first, I must at once admit that I am not able to prove beyond doubt that the milder cases I have adduced are of undoubtedly septic character. A real proof could of course only be adduced in the shape of cultures of pathogenic micro-organisms being obtained from such slighter forms of cedematous inflammation as those observed in my first four cases. This I have not been able to do myself, and it must be left to future observers to adduce such stringent proofs. Already at present, however, I wish from the *clinical* point of view to state that the inflammation in these cases is unlike an ordinary catarrhal one. I am fully aware that an cedematous inflammation can be brought about by other causes than septic ones, that it may be the result of chemical, thermic, and traumatic irritation as well as that it may represent the acme of an intense catarrhal process, but in cases of the three former categories, if met with in

adults, one is almost always able to make out a definite cause for the occurrence of the acute œdematous inflammation; whilst in the instances in which a mere catarrhal inflammation leads to œdema, the latter forms as it were the *climax* of the inflammatory process, and is not reached until after some measureable time. In the cases here under consideration, however, the course of events is quite different. The process *commences*, without any demonstrable immediate cause, with the appearance of an intense œdematous inflammation, the maximum of which, as I have tried repeatedly to point out, usually is reached at its very onset; it further *singles out* certain parts of the upper air-passages, and is not general from its very beginning, as seen in ordinary catarrhal inflammations. Further, the *dimensions* of the œdematous swelling far surpass what is observed in an ordinary catarrhal inflammation; again, the attendant *constitutional* symptoms, shivering, rigor, febrility, and general feeling of malaise, point to a septic origin; and, finally, I wish to point out that even in the slightest of my own cases in several instances definite proof was obtained of the patients having been exposed to *septic* influences, such as sewer gas and drainage poisoning. I fully admit, however, as already stated before, that further bacteriological research must definitely decide whether the slightest forms of œdematous inflammation of the pharynx and larynx are, in reality, due to septic causes.

The second objection which might be raised against my view of the unity of these various processes, namely, that they must differ in kind because they differ in primary localisation, is one which I think it is not difficult to refute. This can best be done, it appears to me, by a simple reference to the analogous case of diphtheria. Diphtheria remains diphtheria, whether it primarily attacks the pharynx, the larynx, the nose, the bronchial tubes, the conjunctiva, the vulva, the rectum, or any wounded surface, and equally so whether it remains limited to the part first affected or whether it spreads to

other parts by contiguity or auto-infection. Obviously it will be most frequently primarily met with in the pharynx, because that part, from its situation, its function as the portal of respiration, and from the histological configuration of some of its constituents, notably of the tonsils, forms the most natural highway for the entrance of pathogenic micro-organisms. In cases, however, in which the pharynx happens to be healthy, whilst the larynx is in a catarrhal condition with some lesion of the epithelium, or in which for any other reason the latter has been injured, it is quite conceivable that these micro-organisms may pass through the pharynx and primarily settle in such parts in which favorable conditions for their further development offer themselves.

It appears to me that the question of the primary localisation of acute septic inflammations of the throat and neck is absolutely analogous to the one just discussed. From theoretical reasons as well as from the experience, not only of myself but also of other observers, the pharynx forms the starting-point of the septic inflammation in the vast majority of cases, and I wish here particularly to draw attention to the rôle played by the anatomical constitution of the tonsils, not only in this but also in other forms of infectious diseases. The epithelial covering of these glands shows gaps large enough to allow a transit of leucocytes, and Philipp Stoeck<sup>1</sup> has drawn attention to the fact that an enormous transit of such cells undoubtedly occurs in the tonsils, without the epithelial strands being actually destroyed. Obviously an anatomical arrangement of this kind fully suffices to explain the predilection with which infectious processes occurring in the mouth localise themselves primarily in the tonsils, and I believe that in a not far distant time these glands will also in a good many cases be held responsible for the entrance of the tubercle bacillus and possibly of other micro-organisms into the system. In

<sup>1</sup> 'Biologisches Centralblatt,' vol. iv, No. 12, und 'Sitzungsbericht der Physikalisch-medizinischen Gesellschaft zu Würzburg,' 1883, No. 6.

some papers recently published in Virchow's 'Archiv' by Emil Schlenker,<sup>1</sup> who worked with Dr. Hanau, and by Emil Krueckmann,<sup>2</sup> it was shown that tuberculosis of the cervical lymphatic glands almost always depends upon the invasion of these glands by the bacillus *from the tonsils*, and in no less than about 60 per cent. of cases of tuberculosis of the lungs examined on the post-mortem table by the last-named observer, tubercles were detected in the tonsils, similar results having been previously obtained by Strassmann and Dmochowski.

That diphtheria preferentially makes its first appearance on the tonsils is too well known a fact to be insisted upon at any length. In the series of cases, too, which I have adduced in this paper, it will have been observed that in a considerable proportion of the cases tonsillitis formed one of the initial symptoms of the septic inflammation.

Facts of this character go far, I think, to explain the different localisation of the septic inflammations of the throat and neck. In the majority of cases their first seat will be the pharynx, and the disease may either remain limited to that part or spread thence; in other cases, however, the micro-organisms may pass through gaps in the epithelial covers of the tonsils or the follicles at the base of the tongue or in the floor of the mouth, and find their way into the cellular tissue of the neck, where, of course, they may also penetrate by other breaches of surface, by carious teeth, &c. In the third class of cases they may with the inspiratory current of air be sucked through the pharynx and be directly inoculated into the larynx, which probably in such instances has become more vulnerable by a previous catarrhal inflammation or by some other cause leading to breaches of surface. Future observers

<sup>1</sup> "Untersuchungen über die Entstehung der Tuberkulose der Halsdrüsen, besonders über ihre Beziehungen zur Tuberkulose der Tonsillen," Virchow's 'Archiv,' vol. cxxxiv, pp. 161 *et seq.*, and 247 *et seq.*, 1893.

<sup>2</sup> "Ueber die Beziehungen der Tuberkulose der Halsdrüsen zu der Tonsillen," Virchow's 'Archiv,' vol. cxxviii, p. 534, 1894.

will have to investigate why in such cases the brunt of the inflammation preferentially falls upon the epiglottis, a part in which the mucous membrane is closely attached to the perichondrium of the cartilage. Enough however, I think, has been shown to render it highly probable that the question of primary localisation after all depends only upon *accidental* circumstances of the individual case, and considering the identity of the clinical and pathological events occurring afterwards, I think it will be generally admitted that no real difference can be established between these septic processes on the mere strength of their having shown themselves in one case first in the pharynx, in another in the larynx, in the third in the cellular tissue of the neck.

To two points I wish to draw attention in conclusion of this part of my subject. It is remarkable that in the cases in which the process begins first in the pharynx, and in which extension occurs, this extension should so decidedly take place in a *downward* direction. It will have been observed that in all my cases the nose and naso-pharyngeal cavity remained unaffected, however intense the inflammation might have been in the close neighbourhood of those parts. I will, of course, not for one moment maintain that this is the universal rule. It is well enough known that erysipelas sometimes primarily appears in the nose, and thence spreads through the naso-pharyngeal cavity into the pharynx and further down, or that it extends from the nose to the external integument of the face; but anyhow it appears to me very remarkable that in all my cases nose and naso-pharynx should have so entirely escaped. The case mentioned previously, however, observed by von Stein, certainly shows that this is not an unexceptional rule, and that an originally pharyngeal septic inflammation may also extend in an upward direction.—Secondly, it may be thought by some rather revolutionary that I venture to include Ludwig's angina amongst those cases in which the septic inflammation originally beyond doubt started from the mucous mem-

brane of the upper air-passages. My reply to this is that the whole existence of this affection as a disease *sui generis* has been by no means fully established; that it was first described at a time (1836) when no examination of the larynx could be made, and that it is already admitted<sup>1</sup> that its ultimate cause consists in the immigration of micro-organisms through some spot of the mucous membrane of the mouth denuded of epithelium. It therefore appears to me not justified by the real facts, to artificially maintain a difference from the mere accident of the primary localisation in the cellular tissue of the neck, whilst the affection itself is pathologically identical with those taking place in the mucosa and submucosa of the respiratory mucous membranes.

The third objection to the view of the unity of the forms of septic inflammations of the throat and neck, namely, that the fever in the different forms takes a different course, is one that can be dismissed, I think, with very few words, and indeed I only mention it particularly because Massei lays great stress upon the fever curve as characteristic for the form of septic inflammation he has described as primary erysipelas of the larynx. It appears to me, however, that the height of the temperature and the fever curve in these cases will so entirely depend (a) upon the quantity and the virulence of the poison which has penetrated into the organism, (b) upon the question whether the resulting inflammation be of an cedematous or of a purulent or of a mixed character, and (c) possibly also upon the question in which tissues it was first localised, that one cannot expect the fever in these cases to take a simply typical form. I can again for the purpose of illustrating what I mean do no better than return to the analogous case of diphtheria. In this disease also the degree of fever varies most markedly—whilst there is never any tendency to extreme hyperpyrexia, the temperature in the severest and most malignant cases is

<sup>1</sup> See, for instance, Schech, 'Die Krankheiten der Mundhöhle, des Rachens und der Nase,' 3rd edition, 1889, p. 81.

often quite low.<sup>1</sup> Yet nobody has ever suggested, so far as I know, that the difference of temperature in various cases of this disease constitutes a difference of kind between them. Further observations, more accurate than I have unfortunately been able to make them in a good many of my cases, will have to show whether any general type of fever after all exists in these septic inflammations, but whatever the results of such an inquiry may be, they cannot influence, it appears to me, our general views as to the identity or non-identity of the individual forms.

The most important objection, however, to my view of the unity of these septic inflammations, and the one which is sure to be raised, is this, that in a number of cases the inflammation was of a purely œdematous, and in another class of an entirely purulent character, and that this fact decidedly pointed to these two classes being of different origin. A few years ago, indeed, this objection would have been simply fatal to my suggestions. It was then dogmatically taught and universally believed that there were two different pathogenic micro-organisms, the one the *Streptococcus pyogenes*, the other one the *Streptococcus erysipelatosus* (Fehleisen's), and that these two micro-organisms, though indistinguishable under the microscope and in pure cultures, yet were essentially different, inasmuch as the former always and without exception produced a purulent, the second equally unexceptionally an œdematous, form of inflammation.

Cases such as my two last would under this teaching of course have been referred to a *mixed* infection, which explanation is at any rate a very convenient one. In the course of the last few years, however, the bacteriological opinion concerning this question has very considerably veered round. Already in 1891 the specificity of Fehleisen's erysipelas coccus had become more and more doubtful, and its real identity with the *Streptococcus pyogenes* more probable. To fully prove this, however,

<sup>1</sup> Morell Mackenzie, 'Diseases of the Throat and Nose,' vol. i, 1880, p. 142.



two more points were wanting, namely, the demonstration in the human subject that the *Streptococcus pyogenes* could also produce erysipelas, and, secondly, the demonstration that also other pyogenic micro-organisms could produce the same result. This proof was definitely given at the meeting of the German Surgical Society in 1891 in an able paper "On the Ætiology of Erysipelas,"<sup>1</sup> read by Dr. Max Jordan, Professor Czerny's assistant at the Surgical Clinique of Heidelberg.

The author was in a position to fill the gap by the clinical observation and bacteriological investigation of two cases of erysipelas, with the result that the question of the nature of the erysipelas coccus was decided in the sense of its non-specificity.

Other facts recorded in literature and quoted by the author, taken in conjunction with the experiences gained in these two cases, enabled him to draw the two following conclusions :

1. Erysipelas ætiologically considered is not a specific disease ; usually it is caused by the *Streptococcus pyogenes*, but it may also be produced by the *Staphylococcus pyogenes aureus*.

2. The micro-organisms causing erysipelas most probably in every case enter into the circulation ; pyæmia following erysipelas is therefore of a primary character, and not due to a mixed infection.

These general conclusions, I need not say, go in exactly the same direction as the propositions of the present paper, and lend, I think, in themselves considerable force to my views on the special subject now under consideration.

But a much more powerful ally of these views has arisen lately in a second paper by Dr. Jordan, in which this author from the broadest conceivable basis dis-

<sup>1</sup> Langenbeck's 'Archiv für klinische Chirurgie,' vol. xlii, 1891, pp. 325 *et seq.* A résumé of this paper has been given by Dr. Jordan himself in the 'Centralblatt für Chirurgie,' vol. xviii, 1891, Appendix, pp. 77 and 78, and my description of his conclusions is a *verbatim* translation of this report.

cusses the nature of septic infections in general.<sup>1</sup> So important, indeed, is this paper with regard to the point I wish to prove, that I must be allowed to give a short *résumé* of those parts of its introduction in which the author in the most lucid and convincing manner summarises the present bacteriological views on the nature of inflammations in general, on the ætiology of suppuration, on bacteria as causes of purulent inflammation, on the mode of action of pyogenic cocci, and on the influence of the nature and condition of the tissues in which the inflammation occurs.

Dr. Jordan follows Cohnheim's teaching with regard to the pathology of inflammation, in starting from the thesis that the "inflammatory irritation" ("Entzündungsreiz") primarily attacks the blood-vessels, causes them to become dilated and produces an alteration of their walls, as a consequence of which augmented transudation and increased permeability for leucocytes occur; the emigration of the latter (diapedesis) is the cardinal symptom. The *intensity* of the inflammatory irritation is of importance in determining the character of the resulting exudation; hence we have to distinguish between serous, fibrinous or croupous, purulent and hæmorrhagic inflammations. From an analysis of experimental investigation concerning the question of suppuration, Jordan then comes—amongst others—to the following conclusions:

1. *No specific pus microbes exist.* Apart from the Staphylococci and Streptococci a large number of other bacteria exist which under certain circumstances can produce a purulent inflammation.

2. *The pyogenic cocci proper are capable of producing apart from purulent inflammation all other forms of inflammation.*

These points are now proven in detail; the author

<sup>1</sup> "Die acute Osteomyelitis und ihr Verhältniss zu den pyogenen Infectionen auf Grund klinisch-bacteriologischer Beobachtungen sowie des jetzigen Zustandes der Bacteriologie," 'Beiträge zur klinischen Chirurgie,' vol. x, Tübingen, 1893.

shows by quotations from the bacteriological work of the most prominent bacteriologists within the last decade that: (1) the *Staphylococcus aureus albus* and *citreus*, (2) the *Streptococcus pyogenes*, (3) the *Staphylococcus citreus albus* and *flavus*, (4) the *Micrococcus pyogenes tenuis*, (5) the *Micrococcus tetragenus*, (6) the *pneumococcus* (Fraenkel und Weichselbaum), (7) the *Bacillus pyogenes foetidus*, (8) the *Bacillus typhosus*, (9) the *Bacterium coli commune*, (10) the *Bacillus pyocyaneus*, can each of them *alone* produce a *purulent* inflammation. In special connection with the subject here under consideration his statement that nowadays the *identity* of the *Streptococcus pyogenes* with the *Streptococcus erysipelatosus* is *fully acknowledged* by the most prominent bacteriologists (Baumgarten, Fraenkel, Cornil, Babes, and others) is of special importance.

The second point, namely, that the action of the pyogenic cocci proper is not an *exclusively* pyogenic one, is then equally exhaustively proved. Jordan shows: (1) that the *Staphylococcus* may produce a purely *serous* inflammation: and (2) that the *Streptococcus* may produce the *most various* forms of inflammation, apart from the purulent one, namely, (a) *serous*, (b) *fibrinous*, and (c) *necrotising* inflammation.

In the following two paragraphs he discusses the mode of action of pyogenic cocci and their pathological dignity, and comes, from actually observed facts, to the conclusion that in a previously perfectly healthy organism, without any concomitant action of predisposing moments, small quantities of pyogenic cocci suffice to produce a purulent reaction of the tissues. This naturally leads to his discussing the conditions under which an infection can take place. He shows that the presence of pyogenic micro-organisms in the system does not lead with absolute necessity to the production of suppuration, and states that in order that the latter may occur two conditions are necessary, namely, first, that a certain *quantity* of germs be introduced, and, secondly, that they must possess a

certain *virulence*. Already Fehleisen had found that *small* doses of pyogenic microbes have no effect upon dogs and rabbits, whilst the injection of *large* doses leads to speedy death of the animals. Similar results were obtained by Watson Cheyne and Hermann. As to the question of virulence, this is a *variable* and not a constant quality; it depends upon a number of different factors, particularly on the condition of *nutrition* and the *age* of the cultures of the micro-organisms.

Finally Jordan shows that the *nature* and the *condition* of the *tissues* invaded by pyogenic organisms may produce considerable differences in the nature of the resulting inflammation. The individual tissues show very different resistance to the invasion of various micro-organisms, and their resistance further varies if any alteration exists in them due to local or constitutional causes. Of particular interest in connection with the question here under consideration is the fact that, as Bujwid's experiments have shown, in cases of *diabetes* the power of resistance of the tissue elements becomes diminished. These experiments were made in order to investigate the cause of the frequency of abscesses and furuncles in diabetic persons, and throw an interesting side-light upon the fact that in several of my own cases sugar was found in the urine during the septic inflammation of the throat and neck, or that the disease itself occurred in persons previously diabetic.

It is obvious that these conclusions of Dr. Jordan's form the most complete and most satisfactory vindication of my position that I could possibly desire. Viewed in the light of his statements, my propositions, revolutionary as they may have appeared from the mere clinical point of view, become in fact *a simple clinical application of general bacteriological principles to a certain group of septic inflammations*. For if it be conceded—as it must be now, I think—that no pyogenic micro-organisms exist which have an exclusive and specific action in producing suppuration, and that various microbes are capable of producing, according to their virulence, their quantity, and the character of

the tissues in which they develop, *any* form of inflammation (*i. e.* serous, fibrinous, purulent, or hæmorrhagic), it is obvious that the *special* form of exudation met with in a given case can no longer be looked upon as the decisive criterion for the pathological classification of the individual case, and that a terminology based upon such non-existing differences must be given up as illogical and erroneous. This is what I have tried to establish in this paper from the purely clinical point of view, and it is most gratifying that the results of an independent clinical study should so entirely agree with the outcome of the accumulated bacteriological teaching of the present day.

I am of course fully aware that the final proof of the correctness of my views must still be adduced. Only when it has been shown that in the slightest as well as in the most severe forms of septic inflammations of the throat and neck pathogenic organisms are met with, cultures of which will in one case produce a serous, in another case a purulent inflammation, will the question have been ultimately solved. I am, however, fully convinced that this can only be a question of time; and I hope that I have succeeded in making it at any rate extremely probable that the various forms of septic inflammations of the throat and neck are in reality pathologically identical.

Should a general agreement to that effect be obtained, the next step will of course be a sweeping revision of our present nomenclature concerning oedematous affections of the larynx. The chapter "Oedema of the Larynx," commonly met with nowadays in text-books of laryngology either ought to disappear completely, as a serous transudation into the tissues of that organ is a symptom caused by the most heterogeneous affections, or at any rate it should be limited according to Kuttner's proposal to the *passive* forms of oedema, such as are met with in Bright's disease. On the other hand, no separate chapters ought to be accorded to "Inflammatory Oedema," "Erysipelas,"

and "Phlegmon of the Pharynx or Larynx," and "Angina Ludovici"; and the conditions hitherto described under these names should be summarised as different stages of one and the same pathological process under the one general heading, "Acute Septic Inflammations of the Throat and Neck."

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. vii, p. 133.)

# MICRO-ORGANISMS IN THE HEALTHY NOSE

BY

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IN initiating a series of researches on the bacteriology of the upper air-passages, we thought it advisable to commence with the nasal cavity, and in the first instance to try and determine the number and characters of any bacteria there met with in health. This would seem only a natural and simple method of procedure, yet it is remarkable that in the large mass of literature we have gone through referring to the bacteriology of the nose, only two articles have been discovered entirely devoted to the normal or physiological condition. One of these is by Dr. Jonathan Wright, to whom we are indebted for many references, and the other is by L. von Besser; both were published in 1889. Dr. Wright's article entitled "Nasal Bacteria in Health" was published in the 'New York Medical Journal' (27th July, 1889), and the author therein comments on the abundance of literature concerning the bacteria of nasal disease, and the entire absence of any systematic examination of normal nasal secretion. Yet

in the literature of the six years which have nearly elapsed since then, we have failed to discover any but the most incidental reference to the subject under consideration. Observers have continued to devote themselves to the bacteriological examination of pathological conditions, thus accumulating a vast amount of recorded work, which we have had to read through in order to extract any previous observations on the micro-organisms of the healthy nose. Yet surely the extensive observations on pathological conditions which have been recorded can have relatively little value until we know the condition of affairs in a state of health. We venture to think also that many contradictory results might have been avoided if the physiological condition of the Schneiderian membrane with regard to microbes had been first determined.

A wider and more important interest is given to the subject when we consider the nose as one of the chief portals in the body for infection. With few exceptions all pathogenic germs enter the body with the food, that is, through the alimentary tract, or with the air, that is, through the respiratory tract. The first stage of the latter being the nose, the bacteriological study of this organ should yield some important knowledge of the first lines of defence of the organism, and indicate how the struggle between the microbe and the cell is carried on.

Taking in chronological order the bibliography we have collected, we find that Hüter in 1873 ('Allgem. Chirurgie,' Leipzig, p. 257) refers to the presence of micrococci in the nose as the true source of irritation in coryza; and in 1876 B. Fränkel ('Ziemssen's Cyclopædia of Medicine,' Eng. edit., art. "Coryza") referring to the same subject writes: "Microscopic examination of the mucous secretion always shows white blood- (pus-) corpuscles, besides the so-called mucous corpuscles, and sometimes red blood-corpuscles and epithelium. A large number of these little structures, recently so much spoken of and called micrococci, may generally be seen also covering the cells." It will be noticed at once that neither of



these observations concerns the quite normal nose; and we have given them simply for their historical interest, as being the first references of any kind to nasal bacteriology. In 1881 J. Herzog ('Wiener med. Presse,' 1881, No. 31, p. 977) said "I have for a long time examined microscopically the nasal secretion from healthy and unhealthy noses, and found in the different secretions besides much detritus, fatty-degenerated epithelium, and a larger or smaller number of pus-corpuscles, no special distinctive appearance in any particular patient. Fungi, micrococci, &c., I found both in healthy and unhealthy secretion, in the latter naturally in larger numbers, and especially so in foetid rhinitis." In 1883, Seifert ('Volkmann's Vorträge,' 1883, "Innere Medicin," 84 (240), s. 2201, Ueber "Influenza,") described the micrococci met with in cases of influenza in the secretions of the respiratory organs—nasal, bronchial, &c. He says (pp. 22 and 23), "Our control researches were extended in the first instance to the nasal secretion of three individuals who were suffering from an ordinary catarrh (cold), and to two individuals who were affected with catarrh following measles. In neither of these cases were micrococci found in the secretion such as were described above in influenza." In an article on ozæna published in 1885 ('Deutsch. med. Woch.,' No. 1, 1885, p. 5, "Die Natur und die Behandlung der Ozæna") Löwenberg made, incidentally, some remark on the micro-organisms of the healthy nose. He said, "The nasal mucus forms a bad nutrient medium for the atmospheric germs. This factor I have established by numerous investigations of the normal nasal secretions, in which I have never found any abundance of fission fungi." His observations were either unnoticed or unconfirmed by subsequent observers, who arrived at very different conclusions. We come first to E. Weibel, who in 1887 ('Centralblatt für Bakter.,' ii, 465) found in the nasal secretion of healthy persons a curved bacillus, which when cultivated in nutrient gelatine and agar develops a spirillum-like body wound in several

coils. In the same year Reimann ('Ueber Micro-organismen im Nasensekret bei Ozæna,' Inaug.-Dissert., Würzburg, 1887; and ref. in Baumgarten's 'Jahresbericht,' vol. iii, 1887, p. 417) regularly found two species of bacteria in the normal secretion, the one a short plump bacillus, the other a small coccus occurring usually in pairs, but often in longer chains; both of these liquefied gelatine, but were not otherwise distinguished by any special culture characteristics.

The view that the nose is a septic depository received further confirmation during this year in the paper read by Rohrer at the 60 Versammlung deutscher Naturforscher und Aerzte in Wiesbaden (ref. in 'Centralblatt für Bakt.,' Bd. iii, 1888, "Bakteriologische Beobachtungen bei Affektionen des Ohres und des Nasen-Rachenraumes.") He remarks: "It is self-evident that the nose and nasopharynx, with its important arrangement of surface for filtering and warming the respired air, must show a large number of fungi. It is well known that besides the microscopic small bacilli and cocci forms, the universally present Hyphomycetes of the aspergillus and mucor groups also deposit their spores in the secretion of the nasal cavity." Parenthetically we would here remark that Rohrer's view that the bacillary richness of the pituitary membrane is "*selbstverständlich*" would appear to represent a wide-spread idea, and shows the retarding effect of taking things for granted. This reaches its most amusing acme in a recent paragraph of the 'British Medical Journal,' where we are solemnly advised to wash the interior of our noses on the extraordinary assumption that "they get quite as dirty as our teeth," and "that the civilised nose is, in fact, one of the dirtiest organs of the body" (26th January, 1895). Possibly the same inspirer of paragraphs is responsible for the further advice given to protect against influenza by drawing an antiseptic solution into the nose ('Brit. Med. Journ.,' 2nd March, 1895). The refutation of these suggestions we will leave until the end of our paper, only remarking here

that the mucous interior of ordinary healthy noses appears to the naked eye, with rare exceptions, to be nearly as clean and as little in need of washing as the conjunctiva. The nasal cavity, as we shall show, seems to be self-cleansing.

We next met with the results of Hajek's observations in 1888 ("Die Bakterien bei der acuten und chronischen Coryza, &c.," 'Berl. klin. Woch.,' No. 33, 1888), in which the reference to the micro-organisms of the normal nose is only made incidentally—as in all previous references—in the course of researches on diseased conditions. He says, "In the normal nasal cavity, where a slight quantity of liquid secretion not rich in cells is found, one only meets with a very small number of bacteria. This is the more astonishing, as our nasal cavity is partly a filter for the inspired air, frequently laden with particles of dust, and here in the natural order of things the settlement of all sorts of bacteria ought to take place. As remarked, however, this is not the case, and evidently for the reason that the normal nasal mucus contains only a few organic substances, and only a few kinds of bacteria find it sufficient as a nourishing substance. Cover-glass preparations of the normal nasal mucus suitably prepared and stained, sometimes show rods or cocci of varying length and thickness; in plate cultivations it is only exceptionally that one meets with a few colonies. Sometimes there appear a few sarcinæ colonies (orange or yellow), sometimes a diplococcus which one finds in the air, or a bacillus which liquefies gelatine and produces a green colour, sometimes also several of the first mentioned kinds. In most cases it is readily recognised that the kinds met with are constant inhabitants of the air."

We come now, in 1889, to the first paper we have met with dealing entirely with the physiological condition; it is that of Jonathan Wright ("Nasal Bacteria in Health," 'New York Med. Journ.,' July 27th, 1889, p. 92), to which we have already referred. He experimented on ten subjects, and found—"in six cases the *Staphylococcus*

*pyogenes*, in three cases the *Micrococcus flavus decedens*, in one case the *Bacillus lactis ærogenes*, in one case the *Penicillium glaucum*, in one case the *Micrococcus cereus flavus*, in one case the *Micrococcus tetragonus*, once in each of three cases different undescribed forms." We would call attention to the points—(i) that the subjects examined are described as fairly normal ones; (ii) that Dr. Wright, while carefully distinguishing the exact species of micro-organisms met with, gives no information as to their abundance or the reverse; and (iii) that in no instance did a culture remain sterile.

The year 1890 would appear to mark high-water level in the recording of variety and abundance of organisms in the healthy nose. The largest and most varied catalogue is that of L. v. Besser ("Ueber die Bakterien der normalen Luftwege," 'Ziegler's Beiträge zur Pathologischen Anatomie und zur allgemeinen Pathologie,' Band vi, Heft 4, p. 331, 1889, ref. in 'Centralblatt f. Bakt.,' Bd. vii, 1890, p. 151).

The several varieties of micro-organisms were settled by microscopical examinations, cultures, and experiments on animals. Testing thirty individuals employed in the bacteriological laboratory for non-pathogenic organisms, he found no less than thirteen varieties, the most frequent being the *Micrococcus liquefaciens albus*, which was met with twenty-two times. For pathogenic organisms he examined fifty-seven men, of whom twenty-eight were convalescents, but the majority were healthy individuals, some of whom were occupied in the laboratory. He found Friedländer's *Bacillus pneumoniæ* twice, the *Streptococcus pyogenes* seven times, the *Staphylococcus pyogenes aureus* fourteen times, and the *Diplococcus pneumoniæ* (Fränkel-Weichselbaum) fourteen times, or once in every four observations! The bacteria were mostly in considerable numbers, and sometimes existing in pure culture. The pathogenic bacteria occurred less frequently in convalescents than in other individuals. According to von Besser's results the nose is a perfect emporium for

micro-organisms, and the dangerous varieties are to be found even in the most healthy subjects. A less astonishing record is made by Paulsen ("Mikro-organismen in der gesunden Nasenhöhle und beim akuten Schnupfen," read on May 3rd, 1890, before the Physiological Society in Kiel, and published in vol. viii of the 'Centralblatt für Bakt.,' 1890, p. 344). His cultures were only sterile in 17·18 per cent. of his experiments, while 53 per cent. showed from ten colonies upwards. However, he only once met with pathogenic micro-organisms—eighty colonies of a streptococcus. He looked in vain for Friedländer's pneumococcus, and never found Fränkel's *Diplococcus pneumoniae* or the *Staphylococcus pyogenes aureus*.

H. von Schrötter and Winkler ('Beitrag zur Pathologie der Coryza,' Wien, 1890) have isolated the *Staphylococcus cereus flavus* and another similar micro-organism, which they designate *albus*, from the nasal discharge in coryza. But von Jaksch appositely points out that the discovery is obviously of no importance unless it can be shown that these forms do not occur in health. This remark applies equally to all nasal affections.

Lermoyez ('Annal. des Maladies de l'Oreille,' &c., Fév., 1891), in 1891, speaks of "the abundance of microbes which inhabit the nose," and talks of the pneumococcus, the diplococcus of Friedländer, the *Streptococcus pyogenes*, the *Staphylococcus albus* and *cereus* as "the habitual visitors of the nasal fossæ." Referring to erysipelas he says, "the streptococcus of Fehleisen is everywhere; Emmerich has demonstrated its presence in the dust of dirty houses. Moreover, the pathogenic agent not only lives around us but also in us; in one out of every five healthy individuals it exists normally in the nasal fossæ." M. Lermoyez, two years afterwards, had evidently modified these views, for recording some observations made with M. Wurtz ('Annal. des Maladies de l'Oreille,' 1893), he says of nasal mucus that, "as a rule, it contained no microbes."

In 1891 Dr. Macintyre, of Glasgow, gave an address

in which he remarked that "if any one take the trouble to scrape a little of the mucus from the lining of the nostril or the mouth in an apparently healthy subject, a great variety of micro-organisms will be found" ('Journal of Laryngology,' vol. v, 1881, p. 222). We have never obtained a sterile culture of mucus from the mouth, and a comparison of the tubes on the table will serve to contrast the profusion of growth in cultures of buccal mucus and the completely sterile condition or scanty growth of nasal mucus. It is possible, however, that Dr. Macintyre may refer to the vestibule, as he speaks of "the lining of the nostril," and further on says, "In the nostril, for example, a considerable number of cocci are always found, large and small, and several bacillary forms are continually present." If by "nostril" he means the cavity of the nose, he falls into the same error as many other writers.

In his manual on 'Bacteria and their Products' (London, W. Scott, 1892), Dr. Sims Woodhead writes: "It has already been mentioned that the *Streptococcus aureus* and *Streptococcus albus* sometimes occur in the mouth, but it would appear that these forms are more frequently met with in the posterior nares and in the cavities of the nose. To their action is supposed to be due the suppuration or festering that almost invariably follows small operations of the mucous membrane of the nostrils, unless the mucous surface is previously prepared by careful antiseptic washing out of the cavities, and by frequent application of antiseptics after the operation has been performed." We shall point out later on that the nostrils are not lined by mucous membrane but by skin; that micro-organisms are comparatively rare in the nasal fossæ; and that surgeons, from the time of Pirogoff ('Grundzüge der Allgem. Kriegschirurgie,' Leipzig, 1864, p. 91) downwards, have observed that, as a rule, wounds of the nasal cavity heal in the most satisfactory manner, without sepsis and with practically no local reaction. In our experience many surgeons make no attempt to render the interior of the

nose aseptic before operation, and in many cases employ no local antiseptic afterwards. They obtain as good results and in as rapid and satisfactory a manner as those who attempt to disinfect the nose on the presumption that it is septic. But, indeed, no lotion sufficiently strong to have any marked germicidal action could be used over such a delicate surface as the Schneiderian membrane; and, as our experiments will show, the mucous lining of the nose is practically automatically aseptic.

Last year M. Löwenberg carried out at the Pasteur Institute a very complete study of the diplococcus of ozæna. He incidentally remarks, "normal nasal mucus shows few organisms" (*Annal. de l'Institut Pasteur*, Mai, 1894, "Le Microbe de l'Ozène"), and thus confirms his experience of ten years previous. In a private letter to one of us he says, "J'ai toujours insisté sur la rareté de micro-organismes dans le mucus nasal normal."

This somewhat lengthy historical introduction will, we trust, be justified from two points of view. Firstly, because it shows the very marked divergence of views as to the presence of micro-organisms in the healthy nose; and, secondly, because it shows that the majority of observers hold the opinion that the nasal fossæ of the healthy are inhabited by numbers of the most various bacteria. Now our researches have led us to the most opposite results, and compel us to formulate conclusions which are extremely striking when contrasted with those of many of the above observers.

Before giving a description of our work, it may be interesting to give a rough estimate of the number of organisms entering the nose. The amount of tidal air being 500 c.c., and the number of respirations averaging 17 per minute, about 500 litres of air are inspired per hour. The number of organisms in the air varies enormously at different times and under different conditions. Parry Laws found an average of 32 organisms (bacteria and moulds) per 10 litres of air. One of us, about this time last year, in bright weather, found an

average of 52 to 164 organisms per 10 litres in some of the main thoroughfares of London. Percy Frankland found 70 organisms per 10 litres in St. Paul's Churchyard.

Taking the lowest estimate, say 30 organisms per 10 litres, we shall have 1500 organisms inhaled into the nose every hour. Obviously this number is constantly exceeded, and it must be a common event for ten times this number, or 7000 organisms, to pass hourly into each side of the nose.

If it be objected that only a portion of the inspired air passes through the nose, the numbers are still considerable. For, assuming that less than half of the inspired air passes through the nose, say 200 litres per hour, and calculating the number of organisms as before, we shall find that 300 to 3000 bacteria and moulds enter each nasal fossa every hour. As Löwenberg points out, the moist walls of the nasal cavity, covered with sticky mucus, resemble a Hesse's tube (for the bacteriological examination of air), and one would expect that a large proportion of the organisms would be arrested in their passage, and would be found in large numbers.

In order to enable Fellows to follow our methods and statistics better, we will at once briefly indicate our results, and afterwards describe the experiments on which our conclusions are based.

The mucous membrane of the healthy nose only exceptionally shows any micro-organisms whatsoever. The interior of the great majority of normal nasal cavities is perfectly aseptic. On the other hand, the vestibules of the nares, the vibrissæ lining them, and all crusts formed there, are generally swarming with bacteria.

The importance of distinguishing between the vestibule and the actual interior of the nose is one of the chief results of our research. In the literature on the subject we have not once seen that this distinction has been made. Most experimenters simply state that they removed some mucus from the interior of the nose; only Jonathan Wright states that it was obtained "from the mucous



membrane covering the turbinated bones and adjacent portions of the septum," and Paulsen writes that after dilating and illuminating the nasal cavity, it was obtained "by scratching the middle and lower turbinals, the septum, and floor ;" but neither of them notes if there was any precaution used to prevent the mucus, or the wire bearing it, from coming in contact with the vibrissæ. That this source of contamination and error is a real one, the details of our researches will demonstrate. Some previous experimenters have obtained the mucus by placing tampons of sterilised cotton wool within the nose. Any one who is both a bacteriologist and a rhinologist will acknowledge the difficulty of introducing and extracting a tampon without its being contaminated with the vibrissæ at the orifice.

Our method was as follows :—Neither the vestibule nor the interior of the nose was in any way disinfected. A nasal speculum was passed through the flame of a Bunsen burner, with this the alæ of the nostril were opened as widely as possible, a sterilised platinum needle was introduced, and a loop of mucus taken from an accessible region, generally from the septum or inferior turbinal, sometimes from the middle turbinal. Great care was taken both in introducing and withdrawing the wire to avoid contact with the speculum or any vibrissæ projecting between its blades. If contact evidently took place, that needleful of mucus was rejected, and the platinum loop was sterilised before being used again. If any contamination had possibly but not positively taken place, a note to that effect was made. The mucus so obtained was used for making a cover-glass preparation which was stained with gentian violet, or for making a surface culture on slanting agar. The latter was kept in the incubator at 35° C. for four to seven days. Other cultivating temperatures and media were tried, but the results were not different. These were the only two methods employed, as our object was not to differentiate the organisms which might be met with, but simply to detect their

presence, and by adopting the same plan in all cases, a uniform comparison could be made. When the vestibule and vibrissæ were examined, the speculum was frequently dispensed with, as sufficient access to this portion could be obtained by simply tilting upwards and backwards the tip of the nose. One of us collected and numbered the specimens, the other examined and reported on the results, without ever knowing from what part of the nose the specimen was obtained. This eliminated the possibility of our being in the least misled by suggestion, and helps to make us the more confident of our results. Needless to say that all the usual precautions of technique were attended to.

First, with regard to the septic condition of the vestibulum naris twenty-seven cultures were made, and not a single one was sterile; in three only a few colonies developed, in three they numbered from ten to twenty, and in twenty-one the growth of micro-organisms was abundant. In many instances the amount of material on the needle was so small as to be invisible. Fourteen cover-glass preparations of the crusts or mucus were made, and in every one organisms were found; in two they were scanty, and in twelve they were abundant. In the two where they showed few organisms there were neither crusts nor mucus, and the needle after being moved against the hairs collected such a small quantity of material that it was necessary to stir it in a drop of sterilised water.

Coming now to the interior of the nose, we made seventy-six cultures in all, and of these sixty-four remained absolutely sterile. In seven there was a scanty growth, in three the colonies were numerous, and in two there were more than twenty colonies; but in none of the twelve cases in which growth took place out of seventy-six tubes could it be called abundant or were the colonies innumerable. We see, therefore, that on an average 84 per cent. of our examinations of the healthy nasal cavity show it to be quite aseptic. Allowing for the chance of contact with the vibrissæ, which

we have found to be a very considerable risk, the occurrence of any germs in the nose must be still more infrequent than our figures indicate. From the nasal mucus thirty cover-glass preparations were made, and twenty-three of them, or 76 per cent., showed no organism whatever; in five a few organisms were found, and in only two were they numerous. In one of these slides there were abundant large cocci with a few bacilli and torulæ; the specimen was taken from a deposit of dust and mucus on the middle turbinal of a student who had been some time in laboratory air on a frosty and sunless day.

Of the cultures taken from the vibrissæ we remarked that three were scanty; we would add that in two of these the specimens were taken from the septal side of the vestibule, where we have noticed the organisms to be less abundant than on the outer side. The other scanty culture was obtained where there was an abundance of mucus entangled in the hairs; we have generally observed fewer germs when there was a plentiful supply of mucus than when the crusts were drier; but whether this is due to mere dilution or to some other cause, we are at present unable to say.

We said that when obvious contamination of the sterilised needle had taken place, it was not used for inoculation. But it not infrequently occurred that in withdrawing the loop of mucus it was difficult to decide whether or no it had come in contact with a projecting hair. In these cases a note of the possibility was always made, and it is to be remarked that in several of them a growth—but a very slight one—took place. In such cases a second or even a third culture was taken from the same spot until it was considered certain that no contact had taken place: almost invariably the latter specimen remained sterile. Thus when experimenting with one subject we took mucus from the vibrissæ and found 80 to 100 white and orange colonies (Experiment 37 d); on attempting to remove some from the septum it was twice thought that just possibly a vibrissa had been

touched; this was at once noted, and three days afterwards one culture showed two orange colonies, and the other two white colonies (Experiments 37 and 37 *a*); in a third attempt it was felt certain that no contact had taken place, and the culture remained quite sterile (Experiment 37 *b*). These notes were not affixed to the test-tubes, which were simply labelled with the number of the experiment, and, as already described, the inoculations were prepared by one of us and the circumstances noted, while the other described the results in complete ignorance of the source or condition under which the mucus was obtained.

One of the statements frequently made about the nasal fossæ is to the effect that they are full of the dust deposited by the inspired air. In the above 150 observations, only on three occasions was there a visible quantity of dust on the nasal mucous membrane (Experiments 84 *b*, 85 *f*, 85 *g*, and 87 *b*). One (Experiment 84 *b*) was taken from the tuber septi where there was a considerable mass of mucus mixed with the dust; it only showed three pin-point colonies. The second (Experiment 85 *g*) also showed a limited number—only seven—of pin-point colonies. A cover-glass preparation from the same subject showed large cocci and a few bacilli and torulæ (Experiment 85 *f*). The third (Experiment 87 *b*) was sterile. These and numerous other observations on pathological conditions show that in the rare cases where dust is visible within the nose, it usually deposits on the anterior portion of the cartilaginous septum or the anterior extremity of the middle turbinal. Our observations on this dust are not sufficiently numerous to justify any conclusions, but it is noteworthy how limited is the number of organisms mixed with it compared with the profusion found in the dust and *débris* of the vestibule.

These experiments were carried out on thirteen different individuals, each of whom yielded a varying portion of specimens. They varied in age from 16 to 36, and on the whole were up to the normal standard of health as regards

the nose. Still, only seven never complained of nose trouble, the other six were patients with slight degrees of catarrh. Of the thirteen, three were females who were always examined in study air; three males were examined in the same atmosphere; and the remaining seven males were examined in the laboratory, where they had frequently spent one or two hours before being examined. Amongst the latter, micro-organisms were met with in a much greater proportion, but we are at present unable to place sufficient data on this subject before the Society to make it worthy of your consideration.

Since the above was written we find that one of the observers we have quoted has further modified his views on the matter. Lermoyez in March of this year writes (*'Annal. des Maladies de l'Oreille, &c.,'* Mars, 1895, p. 226) that, "In spite of the abundance of dust in inspired air, the normal mucus under the microscope shows only an insignificant number of micro-organisms, and when sown in agar and kept at 37° it hardly ever develops." This quite bears out our observations, although we cannot agree with his subsequent remark that normal mucus "contains, however, some pathogenic microbes; there have been found in it staphylococci, streptococci, and even tubercle bacilli." His reference to the latter organism is no doubt in connection with the article published last year by J. Strauss (*"Sur la présence du bacille de la tuberculose dans les cavités nasales de l'homme sain,"* *'Archiv. de med. experiment and d'anatomie path.,'* 6me Année, No. 4, p. 632, Juillet, 1894). This observer found virulent tubercle bacilli in the nasal cavities of nine out of twenty-nine individuals occupied in hospital wards. None of them showed any trace of tuberculosis. Turning to the manner adopted by Professor Strauss for obtaining the mucus, we find that he used sterilised cotton tampons, and in this "collected the dust, the solid particles, the mucus and crusts" of the nasal cavity. This shows once again that previous observers have drawn no distinction between the vestibule of the nose and the cavity proper. In the

light of our experiments, all hitherto recorded observations of micro-organisms in the nasal mucus must be considerably discounted, and nearly all of them will require repeating. The results can only be of interest and value when we know that they concern the pituitary membrane.

The remarkable scarcity of micro-organisms in the Schneiderian membrane attracted our attention very early in the course of these observations, and we cast about to see if any explanation could be found in the route taken by the air in nasal respiration. It had long been taught and is still commonly believed, that the bulk of the respired air passes along the lower part of the nasal cavity, which is hence called the *pars respiratoria* in contradistinction to the upper or *pars olfactoria*. However, the experiments of Paulsen ("Exper. Untersuch. über die Strömung der Luft in der Nasenhöhle," 'Sitzungsberichte d. Kais. Akad. d. Wissensch. zu Wien,' Bd. lxxxv, Ab. 3, 1882) and Kayser ('Archives of Otol.,' vol. xx, 1891) have shown that during inspiration, in the normal nose, the bulk of the air passes along the septum, above the inferior turbinal, describing a semicircle in its course, and extending upwards nearly to the roof of the nose. In experiments made with magnesia, Kayser found that the powder was chiefly deposited on the anterior inferior portion of the septum and on the anterior edge of the middle turbinal. These conclusions have been confirmed by Franke ('Archiv für Laryngol.,' Bd. i, Heft 2, 1893) and Scheff ('Monatsschrift für Ohrenheilk.,' Nos. 10 and 11, 1894). Now the anatomical arrangement prevented us, in the majority of cases, from obtaining mucus from the middle turbinal, but in no case did we neglect to secure a specimen from the septum in as upward and inward a direction as we could reach. From our thirteen subjects forty-nine preparations of mucus were made from the mucous membrane on the septum, and in only eight (or roughly one in six cases) were any microbes seen. Two of these were cases (Experiments 78 a and 37 c) which had been repeatedly examined with negative results, so

that one is inclined to ascribe the occurrence of microbes in some experiments to an error of technique. Another (Experiment 61) showed only one pin-point colony, and one (Experiment 84 *b*) showed only three pin-point colonies, although from a catarrhal subject and mixed with some dust. The fifth (Experiment 62) had been two hours in the laboratory on a foggy day. The sixth (Experiment 77 *h*) is a cover-glass preparation, and is particularly interesting as showing several phagocytes, each containing four to twelve cocci. Of the remaining two, one shows a few cocci in a cover glass preparation, and the other shows a limited culture. Still we see that the percentage of occasions in which the mucus is sterile remains at 83 per cent., the same average as for the mucous membrane generally. From the middle turbinal we obtained twelve specimens, of which four (or one in three) showed micro-organisms. One revealed four or five cocci and diplococci in a cover-glass, the second (Experiment 36 *b*) showed a growth of only two colonies, the third (Experiment 60 *c*) revealed only a pin-point colony, probably due to contamination, and the fourth (Experiment 85 *f*) was a cover-glass preparation of dust and mucus showing a few bacilli and abundant large cocci. It will be seen that, compared with the results obtained from the interior of the nose as a whole, the proportion of sterile observations (66 per cent.) is less. Organisms would appear to occur more frequently on the middle turbinal than in other parts. The results coincide in part with Kayser's powder experiment, but we do not wish to advance them as being positive conclusions, for our observations have not directly borne on this point, and these results were only incidentally noticed when summarising our experiments.

We submit as a summary of our experiments :

(i) That in all bacterioscopic investigations of the nasal fossæ, in all researches as to the action of the nasal mucus, &c., a clear distinction must be made between the vestibule of the nose and the proper mucous cavity. The former is lined with skin, and is furnished with hairs and

with sudoriferous and sebaceous glands ; it is not part of the nose cavity proper, but only leads to it.

(ii) The neglect of this distinction may account for the discrepancies in previous observations on the subject. Contamination with the lining of the vestibule is difficult to avoid even when this source of error has been realised.

(iii) In the dust and crusts of mucus and débris deposited among the vibrissæ of healthy subjects micro-organisms are never absent. They are rarely scanty in number ; as a rule they are abundant.

(iv) On the Schneiderian membrane the reverse is the case. We do not assert that micro-organisms are completely absent ; obviously some must occasionally occur, but under normal conditions they are never plentiful ; they are rarely even numerous, and in more than 80 per cent. of our observations we have failed to find any, and the mucus was completely sterile. These observations were limited to the anterior part of the nose, and, as not more than a fourth of the cavity is accessible to inspection and examination, it is reasonable to conclude that germs would be found still more infrequently in the deeper portions of the fossæ.

(v) The occurrence of pathogenic organisms must be so infrequent that their presence in the pituitary membrane can only be regarded as quite exceptional.

Fellows will perceive that these preliminary investigations raise a large number of interesting problems.

It has been shown that the expired air is almost completely deprived of germs, and we have to consider where and how they are arrested in the air-passages. The experiments of G. Hildebrandt ("Exp. Untersuch. über das Eindringen pathogener Mikroorg. von den Luftwegen und der Lunge aus," 'Beiträge zur pathologischen Anatomie und Physiologie,' Ziegler and Nauwerck, Bd. ii, 1888, p. 421) would tend to prove that the air is freed from all germs before reaching the trachea, but that the surfaces of the trachea and bronchi alone (as in cases of tracheotomy) are not sufficient to filter off the micro-organisms in



inspired air. We have therefore to consider (i) the degree to which this arrest takes place in the vibrissæ; (ii) the proportion which escapes this barrier and is arrested on the pituitary membrane; (iii) if any escape retention in the tortuous passages of the nose; and (iv) the external circumstances and pathological conditions modifying these processes.

Of those deposited on the mucous membrane we have to investigate how they disappear or are expelled from there; and it has to be determined if the mucus exerts a germicidal, an inhibitory, or merely a mechanical action on the micro-organisms.

We have already carried out a number of experiments on these points; they only require extending and confirming before being made public. Our first investigations are so diametrically opposed to those of many observers, and to a common idea of the subject which appears to have been loosely adopted by a large majority of the profession, that we have thought well to make our views known at once.

The practical application and clinical deductions resulting from these investigations we have not discussed. They are sufficiently obvious.

In conclusion, we beg to express our obligations to Professor Crookshank for permitting us to carry out the greater portion of these experiments in his laboratory at King's College.

TABLE OF EXPERIMENTS.

Reference No. and subject of experiment.	Conditions and data.	Point in nose examined.	Culture.	Cover-glass.
11. Male, S.	Normal nose; laboratory air, Oct. 26th, 1894	Crust in vibrissæ	Numerous small yellow colonies of small micrococci.	Numerous organisms; much squamous epithelium; large number of organisms; 5 or 6 species; fine and coarse bacilli; large and small cocci and torulæ.
12. Male, T.	Do.	Crust on right side	Numerous large white colonies	
		Crust on left side	Do.	
14. Male, H.	Normal nose; thick and large vibrissæ; laboratory air, Oct. 26th, 1894	Crust	Numerous colonies	Large number of bacilli, apparently encapsuled; a sarcina.
15. Male, T.	Normal nose; laboratory air, Oct. 26th, 1894	Vibrissæ	Several large white colonies	
16. Male, G.	—	Crust on right side	Copious growth	Any number of cocci and rods.
18. Female, K.	Normal nose; study air, Oct. 30th, 1894	Crust on left side	Numerous colonies	
		Inferior turbinal on right side	—	Micro-organisms abundant. Many torulæ.
		Inferior turbinal on left side	—	
18 a. Do.	Do.	Middle turbinal, right	—	No organisms. Four or 5 cocci and diplo-cocci.
18 b. Do.	Do.	Septum, right	Sterile.	
18 c. Do.	Do.	Inferior turbinal, right	Do.	Few torulæ.
21. Do.	Normal nose; study air, Oct. 31st, 1894	Septum, left	—	
22. Do.	Do.	Inferior turbinal, left	—	No organisms.
23. Do.	Do.	Vibrissæ; no crusts	Innumerable colonies.	
24. Do.	Do.	Septum	Sterile.	No organisms.
28 a. Do.	Normal nose; study air, Nov. 8th, 1894	Inferior turbinal, left; wire touched vibrissæ on withdrawal	Copious white growth.	
28 d. Do.	Do.	Inferior turbinal, left	Sterile.	Sterile.
28 b. Do.	Do.	Inferior turbinal, right	Do.	
28 c. Do.	Do.	Inferior turbinal, right	Do.	

28 e. Do. 32. Male, L. 32 b. Do.	Do. Normal nose; study air, Nov. 10th, 1894 Do.	Vibrissae, left; no crust Middle turbinal, right Dust in vibrissae, left	Over 80 orange colonies. — —	No organisms. Many organisms; bacilli and diplococci; some cocci; few torulae; squamous epi- thelium.
34. Do. 34 a. Do. 34 b. Do. 34 c. Do. 34 d. Do. 34 e. Do.	Normal nose; study air, Nov. 15th, 1894 Do. Do. Do. Do. Do.	Tuber septi, right Tuber septi Septum, left Do. Vibrissae, left Middle turbinal; touched one vibrissae on with- drawing Inferior turbinal Middle turbinal, left Do. Septum, right Do. Middle turbinal and septum, left	Sterile. Do. Do. Do. Over 60 colonies. One white flat colony.	
34 f. Do. 36. Female, A. 36 a. Do. 36 b. Do. 36 c. Do. 36 d. Do. 37. Female, K. 37 a. Do. 37 b. Do. 37 c. Do. 37 d. Do. 37 e. Do. 38. Do. 38 a. Do. 40. Male, L.	Do. Catarrhal subject; study air, Nov. 16th, 1894 Do. Do. Do. Do. Normal nose; study air, Nov. 16th, 1894 Do. Do. Do. Do. Do. Do. Do. Normal nose; study air, Nov. 20th, 1894	Septum, right Crust on vibrissae, right Septum, left; touched vibrissae Septum, right; touched vibrissae Septum, right; no contamination Septum, left Vibrissae Inferior turbinal, left Septum, left Septum, right Do.	Sterile. — — 2 colonies. Sterile. Innumerable colonies. 2 large orange colonies. 2 large white colonies. Sterile. Several white colonies. 50 to 100 white and orange colonies. Sterile. — Sterile.	No organisms. Do. Do. Do. No micro-organisms. Do.

Reference No. and subject of experiment.	Conditions and date.	Point in nose examined.	Culture.	Cover-glass.
40 a	Normal nose; study air, Nov. 20th, 1894	Middle turbinal, right	Sterile.	
40 b	Do.	Middle turbinal and septum, right	Do.	
40 c	Do.	Inferior turbinal and septum, left	Do.	
40 d	Do.	Septal side of right vestibule	Do.	
40 e	Do.	Inferior turbinal and septum, right	5 colonies.	
40 g	Do.	Inferior turbinal, left	Sterile.	
46. Male, L.	Normal nose; study air, Nov. 22nd, 1894	Same spot, but taken without particular care as to avoiding vibrissae	Do.	
46 a. Do.	Do.	Vibrissae on outer side of left vestibule; no mucus or crust	2 colonies.	
46 b. Do.	Do.	Vibrissae on septal side of left vestibule	Innumerable colonies.	
46 c. Do.	Do.	Inferior turbinal, right	Do.	
46 e. Do.	Do.	Septum, right	One pin-point colony.	
46 g. Do.	Do.	Floor of nose	Sterile.	
46 h. Do.	Do.	Septum, right	Do.	
50. Do.	Normal nose; study air, Nov. 27th, 1894	Septum, right	Do.	
50 a. Do.	Do.	Inferior turbinal, right	Do.	
50 b. Do.	Do.	Mucus from vibrissae in right vestibule	15 to 20 colonies.	
50 c. Do.	Do.	Septal side of vestibule on right side; no vibrissae	2 small white colonies.	
60. Do.	Normal nose; in study after 1 hour's exposure to yellow fog. Dec. 4th, 1894	Septum, right	Sterile.	
60 a	Do.	Inferior turbinal, right	Do.	

60 b	Do.	Middle turbinal, right; ? touched	1 colony.
60 c	Do.	Middle turbinal, right	1 pin-point colony.
60 d	Do.	Vibrissae, right vestibule, outer side	Innumerable colonies.
60 e	Do.	Septum and ventricle of nostril, left	Sterile.
60 f	Do.	Ventriculum naris, right	Do.
61, Male, H.	Normal nose; very hairy vestibule. After 1 hour in laboratory air; foggy day, Dec. 4th, 1894	Septum, right	1 pin-point colony.
61 a	Do.	Inferior turbinal, left	Over 50 colonies.
61 b	Do.	Vibrissae, left	Innumerable.
62, Male, G.	Catarrhal, from nasal obstruction. After 2 hours in laboratory; foggy day, Dec. 4th, 1894	Septum, right	Over 30 colonies.
62 a	Do.	Inferior turbinal, left	1 pin-point colony.
62 b	Do.	Do.	Sterile.
63 a, Male, L.	Normal nose; study air, Dec. 11th, 1894	Septum and left ventriculum naris	Do.
70, Male, Hh.	Fairly normal; laboratory air, Dec. 13th, 1894	Septum, right	Do.
70 a	Do.	Inferior turbinal, right	Do.
70 b	Do.	Septum and inferior turbinal, right	Do.
70 c	Do.	Vibrissae, right	14 colonies.
70 d	Do.	Septum, right	—
70 e	Do.	Inferior turbinal, left	—
70 f	Do.	Inferior turbinal, right	—
70 g	Do.	Vibrissae, right	—

No organisms.  
Organisms very scanty; 2  
to 3 encapsuled diplococci,  
and 2 to 3 long bacilli  
forms.  
No micro-organisms.  
Large number of thick ba-  
cilli with rounded ends.  
Breaking down epithelium  
cells.

Reference No. and subject of experiment.	Conditions and date.	Point in nose examined.	Culture.	Cover-glass.
71. Male, S.	Normal nose; laboratory air, Dec. 13th, 1894	Inferior turbinal	Sterile.	No micro-organisms; material abundant; here and there an epithelial cell; many mucous cells. No micro-organisms; abundant material; few epithelial and mucous cells. Material abundant; in parts quite free from micro-organisms; in other parts numerous organisms—bacteria, cocci, and encapsuled diplococci.
71 a	Do.	Septum, right	Do.	
71 b	Do.	Inferior turbinal, right	Do.	
71 c	Do.	Septum, right	Do.	
71 d	Do.	Do.	Do.	
71 e	Do.	Ventriculum naris, right	Do.	
71 f	Do.	Vibrissae, right; mucus abundant	7 colonies.	
71 g	Do.	Septum, right	—	No micro-organisms; material abundant; here and there an epithelial cell; many mucous cells. No micro-organisms; abundant material; few epithelial and mucous cells. Material abundant; in parts quite free from micro-organisms; in other parts numerous organisms—bacteria, cocci, and encapsuled diplococci.
71 h	Do.	Inferior turbinal, right	—	
71 i	Do.	Ventriculum naris, right; ? touched	—	
72. Male, H. Y.	Normal nose; laboratory air, clear day, Dec. 18th, 1894	Septum, left	Sterile.	No micro-organisms; material abundant; here and there an epithelial cell; many mucous cells. No micro-organisms; abundant material; few epithelial and mucous cells. Material abundant; in parts quite free from micro-organisms; in other parts numerous organisms—bacteria, cocci, and encapsuled diplococci.
72 a	Do.	Inferior turbinal, left	Do.	
72 d.	Do.	Vibrissae	Growth, moderate.	
77. Male, B.	Normal nose; study air, Jan. 26th, 1895	Inferior turbinal, left	Sterile.	
77 a	Do.	Septum, left	Do.	
77 b	Do.	Inferior turbinal, left	Do.	
77 c	Do.	Septum, left	Do.	No micro-organisms; material abundant; here and there an epithelial cell; many mucous cells. No micro-organisms; abundant material; few epithelial and mucous cells. Material abundant; in parts quite free from micro-organisms; in other parts numerous organisms—bacteria, cocci, and encapsuled diplococci.
77 d	Do.	Septum, right	Do.	
77 e	Do.	Inferior turbinal, right	Do.	

77 f	Do.	Inferior turbinal, left	—	No organisms; catarrhal cells; plenty of material.
77 g	Do.	Septum, left	—	No organisms; ciliated epithelium.
77 h	Do.	Septum, right	—	Several groups of micrococci, for the most part in cells, 4 to 12 cocci being found in each cell; much material; several squamous cells; several large leucocytic catarrhal cells.
77 i	Do.	Inferior turbinal, right	—	No organisms; material abundant; squamous epithelium and catarrhal cells.
78. Female, B.	Study air; catarrhal subject, Jan. 26th, 1895	Inferior turbinal, left	Sterile.	No organisms; squamous epithelium.
78 a	Do.	Septum, left	10 to 15 colonies.	No organisms; squamous epithelium.
78 b	Do.	Inferior turbinal, left	—	No organisms; squamous epithelium.
78 c	Do.	Septum, left	—	No organisms; squamous epithelium.
79. Male, B.	Study air; catarrhal subject, Jan. 26th, 1895	Do.	Sterile.	No micro-organisms; abundant material; many catarrhal cells
79 a	Do.	Do.	—	No micro-organisms; abundant material; many catarrhal cells
84. Do.	Study air; catarrhal subject, Jan. 29th, 1895	Inferior turbinal, left	2 pin-point colonies.	No micro-organisms; abundant material; many catarrhal cells
84 a	Do.	Septum, left	Sterile.	No organisms; ciliated epithelium; few catarrhal cells.
84 b	Do.	Left tuber septi; alight dust; much mucus	3 pin-point colonies.	No organisms.
84 c	Do.	Middle turbinal and septum, left	Sterile.	Material scanty; 15 to 20 bacilli; 1 diplococcus.
84 d	Do.	Inferior turbinal, left	—	No organisms; ciliated epithelium; few catarrhal cells.
84 f	Do.	Septum, left	—	No organisms.
84 g	Do.	Vibrissae, left	—	Material scanty; 15 to 20 bacilli; 1 diplococcus.

Reference No. and subject of experiment.	Conditions and date.	Point in nose examined.	Culture.	Cover-glass.
85. Male, D.	Laboratory air on frosty and sunless day; normal nose, Jan. 30th, 1895	Inferior turbinal, left	Sterile.	
85 <i>a</i>	Do.	Septum, left; ? touched	1 large white colony.	No organisms; squamous epithelium.
85 <i>b</i>	Do.	Inferior turbinal, left	Sterile.	No micro-organisms; squamous epithelium.
85 <i>d</i>	Do.	Septum, left	—	Abundant large cocci; few bacilli and torulae; squamous cells.
85 <i>e</i>	Do.	Septum, left	—	
85 <i>f</i>	Do.	Dust and mucus on middle turbinal, left	7 pin-point colonies. Innumerable colonies.	Very numerous organisms.
85 <i>g</i>	Do.	Do.	—	
85 <i>i</i>	Do.	Vibrissae, left	Sterile.	
85 <i>j</i>	Do.	Septum, right		
86. Male, L.	Laboratory air; frosty and sunless day; nose normal; marked vibrissae; Jan. 30th, 1895	Inferior turbinal, right	Do.	No organisms.
86 <i>a</i>	Do.	Do.	—	Bacilli in large numbers.
86 <i>c</i>	Do.	Vibrissae, right; no mucus	Innumerable colonies.	
86 <i>d</i>	Do.	Do.	—	
86 <i>e</i>	Do.	Inferior turbinal, right	Sterile.	
87. Male, R.	Study air; frost and fog; nose catarrhal; Feb. 1st, 1895	Septum, right	Do.	Organisms present, scanty.
87 <i>a</i>	Do.	Middle turbinal, right; a little dust with the mucus	Do.	
87 <i>b</i>	Do.	Vibrissae, right	Innumerable colonies.	
87 <i>c</i>	Do.	Vibrissae, right; crust stirred in sterilised H <sub>2</sub> O	—	
87 <i>d</i>	Do.	Inferior turbinal, left	Sterile.	
87 <i>e</i>	Do.	Septum, left	Do.	
87 <i>f</i>	Do.			



87 i	Do.	Vibrissae, left; no crust or mucus; needle stirred in sterilised H <sub>2</sub> O Septum, left	—	Some thick bacilli; some fine filaments (? leptothrix); material scanty. Few cocci; squamous and catarrhal cells; material abundant.
87 k	Do.		—	
87 m 89	Do. Study air; frost and fog; nose catarrhal; Feb. 7th, 1886	Vibrissae, left; septal side Inferior turbinal, right	Innumerable colonies. Sterile.	
89 a 89 b	Do. Do.	Septum, right Vibrissae, right	Do. Innumerable colonies.	

TABLE OF RESULTS.

CRUSTS AND VIBRIÆ.										INTERIOR OF NOSE.						Experiments, not reckoned in results, where vibrations were accidentally touched and culture was	
Culture.				Cover.			Culture.			Cover.							
Few.	Moderate.	Innumerable.	Sterile.	Few.	Abundant.	Sterile.	Colonies. 2-5 5-15 Over 20.	Sterile.	Few.	Numerous.	Absent.	Scanty.	Abundant.				
3	3	21	0	2	0	12	7 3 2	64	5	2	23	7	1				
Total 27				Total 14			Total 76			Total 30			Total 8				
		11, 12, 12, 14, 16, 16, 16 a, 23, 28 a, 34 d, 36 d, 37 d, 46 b, 46 c, 50 c, 50 b, 71 f, 70 c, 73 d			12, 12, 14, 15, 16, 16 a, 32 b, 70 g, 79 a, 84 g, 85 j, 86 e		36 b 37 c 46 e 60 c 61 62 a	18 b, 18 c, 24, 28 a, 28 b, 28 c, 34, 34 a, 34 b, 34 c, 34 f, 36 c, 37 b, 37 c, 40, 40 a, 40 b, 40 c, 40 d, 40 g, 46, 46 g, 46 h, 50, 50 a, 60, 60 a, 60 e, 60 f, 62 b, 63 a, 70, 70 a, 70 b, 71, 71 a, 71 b, 71 c, 71 d, 71 e, 72, 72 a, 77, 77 a, 77 b, 77 c, 77 d, 77 e, 78, 79, 84 a, 84 c, 85, 85 b, 85 e, 86, 86 a, 87, 87 a, 87 b, 87 e, 87 f, 89, 89 a	18 a 21 63 a 70 e 77 h 77 f, 77 g, 77 i, 78 b, 78 c, 84 e, 84 f, 86 d, 87 h, 85 f, 85 e, 86 e		34 e, 37, 37 a, 46 a, 60 b, 71 i, 85 a	28 a					

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,'  
Third Series, vol. vii, p. 163.)

FURTHER OBSERVATIONS  
ON THE  
DEVELOPMENT OF MAMMARY FUNCTIONS  
BY THE  
SKIN OF LYING-IN WOMEN.

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Received March 26th—Read June 11th, 1895.

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IN vol. lxi, p. 419, of the 'Medico-Chirurgical Transactions' will be found a paper "On the Development of Mammary Functions by the Skin of Lying-in Women," with a record of thirty cases (p. 438).

The description of the appearances was given as follows (p. 422):

"1. They are situated in the skin of the axilla, which cannot be pinched up freely over them. On attempting to raise the skin it seems to be tied to the lumps by fibrous septa.

"2. They can be raised and isolated from the deeper  
VOL. LXXVIII.

structures, and are not in the situation or of the shape and feeling of glands.

"3. The skin over them is usually quite natural in appearance.

"4. They are limited to the hair-covered surface.

"5. They are usually soft and somewhat elastic, except when swollen.

"6. They are usually somewhat flattened, their vertical diameter being the smallest.

"7. They do not possess any nipple, pore, or duct.

"8. Their size varies from the smallest perceptible to that of an egg, or perhaps larger."

As regards their course, no quotation need here be made.

As regards their secretion—

"1. In the first eleven the mode of obtaining the secretion had not been discovered.

"2. In the remaining nineteen (with one exception) secretion of some fluid was obtained. . . .

"5. The secretion was of three principal kinds:—(a) Granular debris, like the secretion of sebaceous follicles; (b) colostrum; (c) milk. . . .

"8. At the same time various follicles (in the skin over the lump) would produce various secretions. . . .

"9. The secretion was expressed from the situation of the sebaceous follicles, as marked by the situation of the hairs."

These lumps were without nipples or pores.

Cases were quoted of other lumps in the axilla, by way of differential description, and to show that the author had not overlooked the following conditions:

A. Extension of *mammæ* into *axillæ* (p. 439).

B. Separate axillary *mammæ* with axillary nipples, pores, or ducts (p. 430).

C. Supernumerary nipples (without special gland-substance) (p. 432).

The following opinion was offered as to their nature (p. 435):

"The cases which I have described, and which I believe have not been hitherto recognised, seem to prove that in lying-in women the sebaceous follicles of the skin are capable of producing true mammary secretions. The transition from granular material, through colostrum, to true milk, is distinct and unmistakable. They confirm the opinion that the breast is a highly specialised aggregation of highly specialised sebaceous follicles. . . .

"I have not yet had an opportunity of making a microscopical examination, but these structures are so far from rare that, when attention is once directed to them, opportunities are sure to arise sooner or later. The secretions were too scanty for chemical analysis. It is far from improbable that they may share the pathological affections of the breast, and even be the seat of abscess."

Since writing the above I have obtained the following material, which we have utilised for microscopical examination :

1. Skin from both axillæ (including subjacent tissue forming distinct lumps) obtained from a woman aged forty-two, who died in St. George's Hospital, having been delivered seven days previously of a six months fœtus.

2. Similar specimens from a woman under the care of Dr. Boxall, who died of uterine hæmorrhage. The autopsy was made twenty-eight hours after death ; old quiescent lung disease was found.

3. A similar case in all respects ; autopsy sixty-three hours after death.

All the specimens were hardened in spirit prior to examination. Sections were cut with a freezing microtome and stained in hæmatoxylin.

Microscopical examination of the "lumps" shows that the structure of each is the same. They consist of (a) a covering of normal skin, (b) loose subcutaneous tissue and fat, (c) aggregations of glands.

With regard to the latter, it must first be pointed out that they are placed at a considerable depth from the surface, in the situation of the sweat glands, and conse-

quently at a much lower level than that of the sebaceous glands.

The glandular tissue in all of the specimens is of the tubular type. It consists of a series of ducts opening through an interpapillary process, each duct passing in a straight or slightly wavy line through the epidermis and corium, and of convoluted tubes with which the ducts are continuous. The epithelium lining the duct is polyhedral, and consists of two or three layers of cells; that lining the deeper part of the tubes is not so well shown, as the specimens have not kept very well in the spirit, but the cells appear to be more columnar. It is, indeed, quite evident that the glands are sweat-glands, but on comparing them with sections of skin from the normal axilla some slight variations are seen.

In the first place, the amount of gland-tissue is greater than in natural skin, and the glands extend to a considerably greater depth from the surface, conditions which are well seen both in the sections and in the drawings from them (Plates IV and V).

In the second place, the arrangement of the glands is much more irregular than in normal skin: they do not seem laid down on so fixed a plan, and the coiled deeper parts are placed at very irregular intervals.

There is no alteration in the cells themselves, and no appearance of any secretion. The sebaceous glands are normal, and may be seen of perfectly natural size in the sections through the lumps. There is no appearance of any mammary tissue or of any other glandular structure beside those already mentioned. In all parts of the coiled tubes involuntary muscle-fibres are abundant, the layer of muscular tissue being more evident than in sections of normal skin.

It will be seen from the foregoing description that the microscopical examination of these lumps did not confirm the original opinion that the axillary lumps in question would prove to be of the nature of sebaceous glands. Indeed, they seem to agree with the description of the

skin-glands in the human axilla by Dr. Creighton in his paper entitled "Three Cases of Tumour arising from Skin-glands in the Dog," published in vol. lxxv of the 'Med.-Chir. Transactions,' from which we quote the following extract.

"It is well known that skin-glands, the same as those of the dog, are found not only in other mammals and in some batrachians, but also in the human axilla, the areola of the breast, the groin, &c. The references made to the axillary skin-glands in recent histological works are apt to convey the impression that the glands are embedded in, or in close connection with, the corium like the ordinary glomerular sweat-glands. But their most characteristic and distinctive occurrence in the axilla is not in the substance of the skin, but as a separate and circumscribed layer, of brownish colour and lobulated surface, adhering to the under surface of the skin. This glandular layer will most probably be met with at the deepest part of the cutaneous recess, over the area where the skin adheres most closely to the axillary fascia. In a large number of observations made in the dissecting-room I have found the glandular structure sometimes as large as a florin and as thick, but more often reduced to a few scattered small lobules, apt to be mistaken for fat lobules, and not unfrequently wanting altogether. In a microscopic section, the tubular coils are found to be separated from one another and enclosed in a large quantity of connective tissue. Good specimens of plain muscular fibres may be obtained with great ease by teasing a minute portion of the glandular structure. These observations for the most part agree with the statement of Sappey,<sup>1</sup> who speaks of the skin-glands of the axilla as being collected into a circular layer under the skin, three to four centimètres in diameter and two millimètres in thickness. They are described also by Kölliker<sup>2</sup> as forming a continuous layer under the skin,

<sup>1</sup> Sappey, 'Traité d'Anatomie descriptive,' vol. iii, p. 505.

<sup>2</sup> Kölliker, 'Handbuch der Gewebelehre,' 1852, p. 147.

and by Frey<sup>1</sup> as being crowded together in a distinct stratum.

"The axillary glandular stratum in man is so variously developed, and so often wanting altogether, that it may be regarded as a rudimentary organ of cutaneous secretion.

"The axillary odour which characterises certain individuals of both sexes is probably associated with the presence of a well-developed stratum of the glandular substance.

\*       \*       \*       \*       \*       \*

"According to an observation of Sappey's,<sup>2</sup> the large axillary skin-glands in man sometimes extend to the lateral and anterior regions of the thorax; and I have in one instance found perfect examples of them in intimate association with the breast structure. The case was that of a woman aged thirty-eight, who had a large soft cancerous tumour removed from the outer part of the mammary region by Mr. Le Gros Clark, at St. Thomas's Hospital, in 1873. I made microscopic sections of the tumour as well as of the structure adjoining it; and in the latter I found several minute cysts, about the size of a pin's head, which proved to be dilatations of the peculiar axillary skin-glands, and were so labelled by me at the time. The walls of each cyst showed, in the most exquisite form, a considerable expanse of plain muscular fibres in close parallel order; a portion of that muscular-fibre membrane is drawn in fig. 2 (Pl. III), and the characteristic large-sized epithelium seated directly on the muscular coat is shown in fig. 3. These glands were in close proximity to ordinary lobules of the breast, but there was little difficulty in distinguishing the one kind of structure from the other."

It will be seen from the foregoing description that the lumps described by us are, in all probability, an enlargement of structures which are normally found in the human

<sup>1</sup> Frey, 'Handbuch der Histologie,' 1870, p. 596.

<sup>2</sup> Quoted by Henle, 'Handbuch der Anatomie des Menschen,' vol. ii, p. 35.



axilla, and that their origin is not to be traced to any aberrant mammary nodules.

The fact that various mammary secretions are produced by the lumps in question, having the characters of sweat-glands, as well as by mammary tissue, would seem to show either that mammary tissue is not necessarily a direct modification of sebaceous gland-tissue, or that similar secretions can be produced by more than one variety of skin-gland. In this connection it may be remembered that the ceruminous glands of the ear, which secrete "wax," are coiled-tubular, and not acinous in structure.

It may further be pointed out that, from the point of view of development, there is nothing incongruous in this assertion, seeing that the mammary structure originates by an ingrowth of the surface epithelium, as do both sudoriparous and sebaceous glands.

The very specialised form of sweat-glands described by Dr. Creighton as occurring in the human axilla, however, forbids the inference that sweat-glands of the ordinary kind are necessarily capable of producing mammary secretions in all parts of the body. Indeed, it is probable that the assumption of mammary functions by skin-glands is limited to these highly specialised sweat-glands.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. vii, p. 175.)

## DESCRIPTION OF PLATES IV AND V.

Further Observations on the Development of Mammary Functions  
by the Skin of Lying-in Women (Dr. FRANCIS HENRY  
CHAMPNEYS, and Mr. ANTHONY A. BOWLBY).

### PLATE IV.

FIG. 1.—Parts of a section through an “axillary lump” and superjacent skin, showing the large irregular masses of specialised sweat-glands extending to a much greater depth than in the normal axilla. Unaltered hair-follicles with their sebaceous glands are seen.

### PLATE V.

FIG. 2.—A portion of “axillary lump” indicated by enclosure in Fig. 1, shown under a higher power.

FIG. 3.—Section through the skin and subcutaneous tissue of normal human axilla, showing the sweat-glands and their ducts.

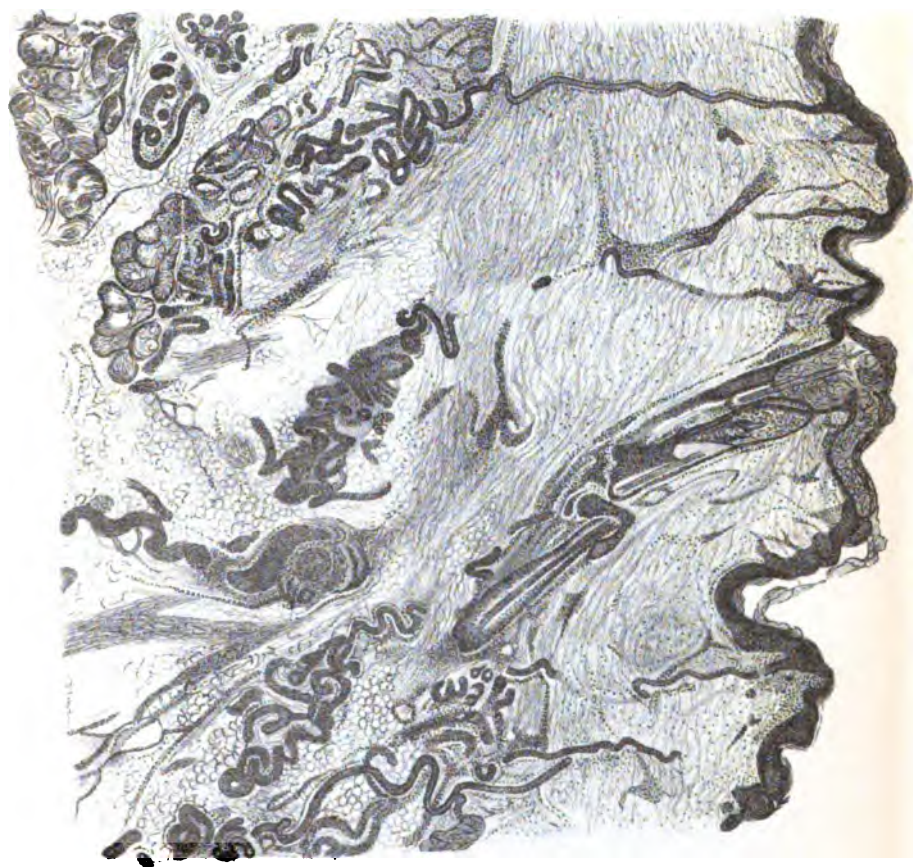
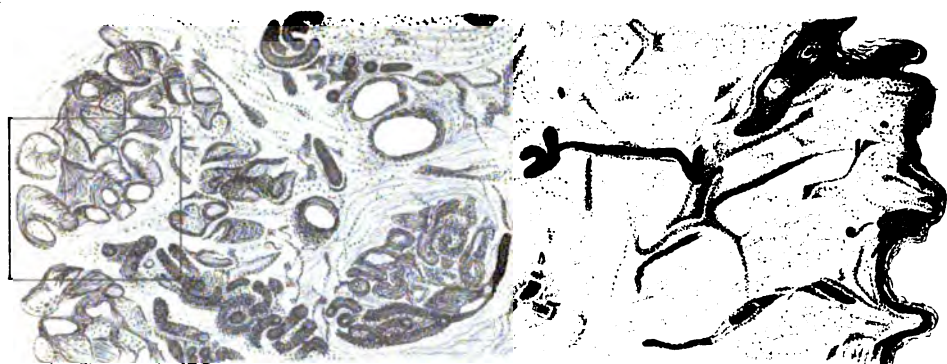
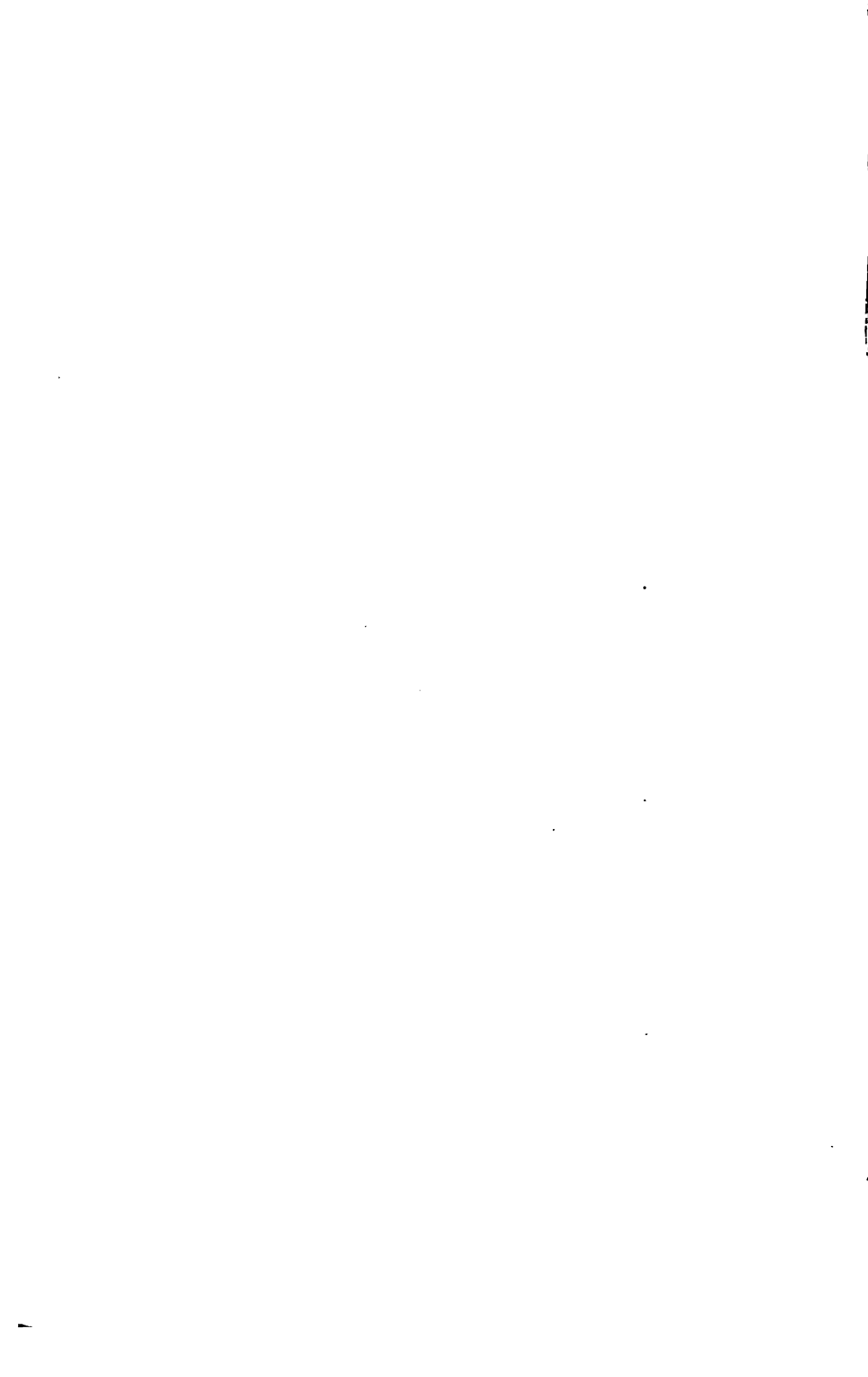


Fig 1



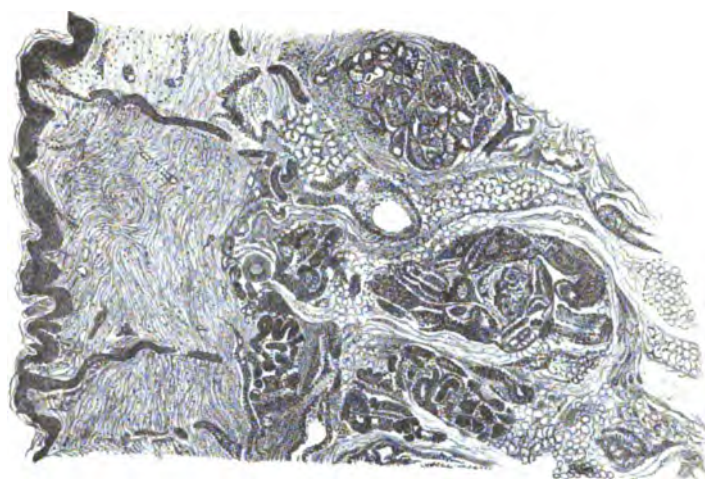


FIG 3.



FIG 2.



A CASE  
OF  
INTRA-PERITONEAL RUPTURE OF THE  
BLADDER;  
SUTURE; RECOVERY.  
WITH REMARKS ON THE INFLATION TEST.

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WITH A TABLE OF TWENTY CASES OF RUPTURE  
OF THE BLADDER TREATED BY SUTURE  
SINCE 1888.

BY  
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Received May 14th—Read June 11th, 1895.

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IN 1888 I had the honour of reading before the Fellows of this Society a case of intra-peritoneal rupture of the bladder treated by suture, and supplemented my paper by a table of eleven cases, which were all the instances of that injury I could at the time find published, in which the same treatment had been adopted. I now venture to bring before the Society a second case, and I do so more

particularly to call attention to a test adopted as a means of diagnosis, namely, inflation with air—a test which I think it will be conceded is one of great value, and one that should be employed in all doubtful cases. My friend Mr. W. Ernest Miles, formerly house surgeon to the Metropolitan Hospital, has been at some trouble to collect the cases of intra-peritoneal rupture, treated by suture, recorded in medical literature since the publication of my table in the Society's 'Transactions' for 1888. These cases, which number seventeen, make, with the eleven published in my former paper, in all twenty-eight treated in this way. For the notes of the case I am indebted to Mr. Miles and to Mr. Meakin, also late house surgeon to the hospital.

V. B—, æt. 42, tailor, was admitted into the Metropolitan Hospital on March 8th at 1 p.m., with a history of having sustained an injury to the lower part of his abdomen.

*History.*—On March 7th, at 6 p.m., the patient, whilst endeavouring to separate two men who were fighting, was knocked down. One of the men fell upon him, striking with his buttocks the lower part of the patient's abdomen as he lay upon his back on the pavement.

Immediately after the injury the patient felt acute pain in the hypogastrium, and thinks that he became unconscious. He was taken home and put to bed, and slept for an hour and a half. On waking, he still felt great pain, and wished to micturate, but failed on attempting to do so. He then sent for Mr. Jackman, who passed a soft rubber catheter, and drew off a pint of urine tinged with blood. Though more comfortable for a time, he could not sleep for pain, and passed as he termed it a "terrible night." In the morning, a large quantity of urine, intimately mixed with bright red blood, together with some blood-clots, was again drawn off. Later in the day he presented himself at the hospital. When questioned as to when he last passed water before the injury, he gave various answers. At first he said at



dinner time (1 p.m.); then he said two hours before the accident; then one hour before. This uncertainty in regard to the time of last passing water, coupled with the fact that he was able to sleep after the injury in spite of great pain, rendered the assumption probable that he was drunk at the time, and had not emptied his bladder for some considerable period before the accident. Since his convalescence he has admitted that this was the case.

*Past History.*—He has always been a heavy drinker, getting drunk frequently. As a rule he rises two or three times during the night to pass water. In other respects he has enjoyed good health.

*On admission.*—When brought into the surgery the patient looked pale, and his face wore an anxious expression. He complained of great pain in the lower part of the abdomen. The pulse was small, quick, and easily compressible. Acute tenderness on pressure was elicited over the hypogastrium. There was no distension at this region, and no dulness to percussion. There was no bulging of the flanks, but some degree of dulness which was equal on both sides. There was no side-to-side fluctuation. The abdominal wall above the umbilicus moved with respiration, but below the latter point it was rigid and motionless, and the abdominal muscles here were hard and resistant. As soon as Mr. Walsham arrived, a silver catheter (No. 10) was passed. On reaching what was taken to be the region of the prostate, a peculiar soft grating was felt, and as the point passed beyond this, blood-stained urine began to flow. The stream increased in volume during inspiration, and diminished during expiration. On withdrawing the catheter slightly the flow stopped, and began again on pressing it further in. About a pint of blood-stained urine was thus withdrawn, after which the patient felt much easier. On depressing the handle the point of the catheter could not be distinctly felt through the anterior abdominal wall. Fourteen ounces of warm 2 per cent. solution of boracic acid were then introduced through the

catheter, of which ten ounces returned. The test was repeated, and seventeen ounces were thrown in, of which ten ounces again returned, but by introducing the catheter to its full extent eight additional ounces were drawn off, which gave one ounce more than had been thrown in on the second occasion.

Since, however, four ounces of the first quantity had remained, there were still three ounces to be accounted for. The evidence by this means was not thought to be sufficiently conclusive of there being a rupture of the bladder, as some of the fluid had escaped at the spot where the rubber tube was fixed to the catheter, and all of the urine might not have been withdrawn the first time. It was decided, therefore, to try the inflation test. For this purpose the india-rubber apparatus belonging to an ether-freezing microtome was utilised, the tube of which was attached to the free end of the catheter. The liver dulness having been carefully percussed out, a few cubic inches of air were forced through the catheter by two or three contractions of the rubber ball. The effect was instantaneous. The abdominal cavity became distended, the liver dulness immediately effaced, and the whole abdomen tympanitic to percussion. The patient fell into a condition closely resembling collapse, he complained of great pain, his respiration was laboured, and the action of the heart turbulent. The diagnosis of ruptured bladder was now placed beyond a doubt, and the patient was at once taken to the operating theatre.

Under chloroform an incision was made in the middle line from the umbilicus to the pubes. On opening the peritoneum a rush of air escaped together with a small quantity of blood-stained fluid. The intestines having been pressed back by means of a large flat sponge, the bladder was found collapsed, with a vertical rent in its posterior wall extending from the summit along the middle line to the base of the trigone bordering on, but not involving the reflection of peritoneum. A silver catheter, when introduced into the bladder, readily passed

into the abdominal cavity through the rent. The coils of small intestine that occupied the recto-vesical pouch were somewhat reddened, but no peritonitis or effused lymph was present. Fourteen Lembert sutures in all were introduced. Beginning as deep down the pelvis as possible eleven interrupted silk sutures were passed by the aid of Hagedorn's needle-holder. The introduction of the finger into the bladder whilst the lowermost sutures were being inserted greatly facilitated the procedure. The uppermost suture was passed well above the upper limit of the rent. These sutures having been tied, a much clearer view than had hitherto been possible of the deepest part of the wound was obtainable, and it was seen that about one third of an inch of the rent in this situation had not been closed. It was impossible to reach and secure this with the Hagedorn or any other holder, but the difficulty was finally overcome by using a Smith's rectangular cleft palate needle, which answered admirably. Three additional sutures were thus passed, and completely closed the rent. Four ounces of milk were then introduced into the bladder through a catheter at some degree of pressure, with the result that the bladder became distended without a single drop exuding through the line of sutures. The pelvic cavity below the sponge keeping back the intestine was then irrigated with 1 in 3000 solution of perchloride of mercury and afterwards with a 2 per cent. solution of boracic acid. The sponge was removed, the pelvis once more irrigated, the abdominal wound closed by first uniting the peritoneum and muscle by buried aseptic silk sutures, and the skin by interrupted sutures, and a collodion dressing finally applied.

The patient suffered very little shock, and about an hour and a half after the operation passed urine spontaneously under him. Subsequently a soft rubber catheter was introduced every four hours of the first twenty-four, after which period the patient was made to pass urine naturally at four-hourly intervals, he being awakened for the purpose if necessary.

From the time of operation the patient may be said to have made an uninterrupted recovery, and it is not necessary to detail the daily notes, which, however, were accurately kept by Mr. Meakin.

*Remarks.*—As far as I have been able to ascertain with the help of Mr. Miles, this is the first case of ruptured bladder in which the "inflation test" has been employed. In January, 1890, Dr. Morton contributed a paper to the 'Journal of the American Medical Association' in which he suggested the use of hydrogen gas for inflating the bladder in cases in which the existence of a rupture in the wall of the bladder was doubtful. He carried out several experiments upon the cadaver, and found that when hydrogen gas was introduced, at moderate pressure, into a bladder that was intact, a localised tympanitic swelling made its appearance in the hypogastrium; but, on the other hand, that if a rent was previously made in the wall of the bladder covered by peritoneum, the whole abdomen at once became tympanitic and the liver dulness was completely lost. Dr. Keen also published a paper in the 'Annals of Surgery' in 1890 in which he advocated the inflation of the bladder, in cases of rupture of that organ, with filtered air. Though to both of these surgeons is due the credit of having suggested this method of diagnosis, yet to neither of them had an opportunity occurred up to the time of publication of their papers for testing the practical value of their suggestions upon the living subject, and I have not been able to ascertain whether the test has been put into actual practice either by them or by any other surgeons since. In the case under consideration the injection of a few cubic inches of air, forced into the bladder by two or three contractions of the rubber hand-ball apparatus, caused the whole abdomen immediately to become markedly distended, and the liver dulness, which had been previously percussed out, to be entirely lost. The insufflation of this small amount of air produced such an instantaneous alteration in the abdominal contour that no

question existed in the minds of any present as to the bladder being ruptured. The patient's condition incidental to the presence of free air in the abdominal cavity is worthy of note. Thus he became greatly distressed, complained of great pain, and experienced a sensation of impending suffocation. His pulse became small, rapid, soft, and easily compressible, and his respiration short and rapid. The immediate improvement in the patient's general condition attending the escape of gas on opening the abdomen shows that these subjective symptoms were entirely due to the presence of free gas in the abdominal cavity. The conclusions drawn from this single experience of the inflation test are briefly these, viz.:—that the amount of air to be introduced need only be very small, not more than three or four cubic inches; that only very moderate pressure is required for the purpose; that the presence of quite a small amount of free gas in the abdominal cavity is sufficient to establish the diagnosis beyond a doubt; and lastly that the introduction of gas into the abdominal cavity even in small quantity is attended by a profound disturbance of the patient's general condition.

With regard to the last circumstance, it might be well on future occasions that the inflation test should be used when the patient is on the operating table, and not when he is in the ward as was the case here, so that in the event of untoward symptoms attending the presence of the gas in the abdominal cavity, immediate relief might be afforded by opening the abdomen. Before the inflation test was applied, I was doubtful whether a rupture of the bladder really existed, and my colleague, Mr. F. C. Wallis, who was present, considered that the signs were hardly sufficiently diagnostic to justify opening the abdomen. For the bladder being collapsed and the rent extending nearly to the prostate, the point of the catheter passed at once, without imparting any sensation of having first entered the bladder, through the rent into the rectovesical pouch, from which about a pint of blood-stained

urine flowed. It is true that a peculiar grating sensation was felt as the catheter passed through the rent, but the situation of this was so near the prostate that the grating sensation appeared, both to Mr. Wallis and myself, to be located in the prostatic urethra. Further, when the point of the catheter had passed through the rent into the recto-vesical pouch, a good deal of lateral movement of the beak of the catheter was obtained, which circumstance inclined us to believe it was in a distended bladder. Moreover, so soon as the urine began to flow away the lateral movement became limited, and this led us still more to believe that the bladder was contracting upon the catheter. This limitation of movement was no doubt due to the intestines falling into the recto-vesical pouch, and replacing the urine that had collected in that situation. The character of the flow of urine from the catheter was noteworthy, in that it varied with respiration, but this of course may happen in conditions other than rupture.

The water-injection test was by no means conclusive. On the first injection, although fourteen ounces were thrown in, only ten returned. Since some leakage had taken place at the point where the rubber tube was fixed to the catheter, it was felt that this loss might account for the disparity above mentioned. Accordingly the experiment was repeated, and seventeen ounces were thrown in, all leakage being stopped; eighteen ounces returned, showing an increase of one ounce. Perhaps more stress might have been laid upon the latter observation than was done at the time. Some surgeons rely upon the water injection test as a crucial one, but in their cases the difference between the amounts injected and returned was so great, as to leave no doubt in their minds that some of the retained fluid had found its way into the peritoneal cavity. In a case of suspected rupture of the bladder that came under my care some years ago, I applied this water test and made several injections, but since exactly the same amount returned as was thrown in,

it was concluded that no rupture existed, and this conjecture subsequently turned out to be correct.

Both tests, therefore, are undoubtedly of value, the water test especially so in those cases in which the abdomen is already tympanitic, and in which, therefore, the inflation test, it has been suggested, might not be conclusive. I would submit that both tests be applied, and I venture to think that with their combined aid the question of rupture in all doubtful cases may at once be set at rest.

The suturing of the rent was attended with much difficulty owing to the great depth of its lower limits.

No form of needle-holder was found suitable for introducing Lembert's sutures at this part, though Hagedorn's served the purpose admirably for suturing the upper three-fourths. When those sutures that had been introduced were tied, a clearer view of the lowermost limit of the rent was obtained, and it was then found that quite a fifth of its entire extent remained unclosed.

After various attempts this part was at length secured by means of a Smith's rectangular cleft-palate needle. Especial attention is called to this because it is from failing to efficiently close the deepest part of the rent that several fatal results have ensued.

As regards the after-treatment of the case, the only point that seems worth discussing is the expediency of tying a catheter in the bladder. Some surgeons advocate this practice on a plea that adequate rest is ensured to the bladder for the proper union of the rent. I would submit that the retention of a catheter can only be attended with danger in that it subjects the patient to the risk of septic infection and cystitis. In my former paper I maintained that it was quite unnecessary if the patient was made to pass water at regular and such intervals as would ensure no danger of over-distension. Further experience tends to confirm this view. In the present case a catheter was only passed five times after the operation, the patient subsequently micturating without its aid every four hours.

In conclusion I would offer a few brief remarks on the facts that I think are brought out by a review of the cases of intra-peritoneal rupture collected by Mr. Miles and myself. Mr. Miles has been able to find only twenty cases published since my paper in 1888. These added to the fourteen cases in my table make a total of thirty-four. In twenty-eight of these thirty-four the rent in the bladder was closed by suture. In the remaining six the bladder was drained either through the wound in the abdominal wall or through an incision in the perineum, the bladder wound itself being either stitched to the abdominal walls or left free, and the peritoneal wound drained or plugged with iodoform gauze. In one of these cases the rent in the bladder was said to be inaccessible to suture.

Of the twenty-eight cases in which sutures were employed eleven recovered and seventeen died.

The cause of death in the seventeen fatal cases, was in ten, peritonitis; in three, shock; in one, shock and peritonitis, the patient dying before the completion of the operation; and in two, shock and hæmorrhage combined, the cause of the hæmorrhage being in one case a tear in the superior vesical veins, and in the other an incision in the perineum made for the purpose of drainage. In one the cause of death is not stated, but from the context it appears probable that it was peritonitis.

It is noteworthy that in the eleven cases that recovered in only one was peritonitis present at the time of operation, whilst conversely of the seventeen that died in eight (and probably nine) acute peritonitis had already set in, showing very clearly the importance of an early diagnosis and operation. At the same time the considerable period that in some of these cases had elapsed before the operation without peritonitis occurring (as much as forty-two, fifty-four, and sixty hours) also shows how long healthy urine may remain in the peritoneum without doing any harm. This fact must not be presumed on, however, since in one case peritonitis had already set in as early as ten hours



after the injury. In the eight or probably nine cases in which peritonitis was present at the time of operation seven and probably eight appear to have died of peritonitis, and one died from shock on the operating table.

In the eight fatal cases in which there was no peritonitis at the time of operation, death was due in five to shock or hæmorrhage or the two combined, and in three to peritonitis subsequent to the operation. In two of the latter cases, the peritonitis was due to leakage of the bladder; in the third the cause of the peritonitis is not stated.

Leakage of the bladder from inefficient suture, or the giving way of a stitch, it will be seen from the combined tables occurred in no less than four of the twenty-eight cases, and in two as mentioned above was the direct cause of peritonitis and consequent fatal result. In the remaining two, peritonitis in one was already present; in the other, death occurred from shock seven hours after the operation, and before the leakage had had time to take effect. It is worth noting that in three of these cases the leakage is said to have occurred at the lower angle of the wound, the spot which in my case was with the greatest difficulty closed, and which could not have been closed without the aid of the rectangular palate-needle.

The importance of testing the competency of the bladder at the conclusion of the suturing by injecting with milk or other equally bland and easily detectable fluid cannot, therefore, be too strongly urged.

*Abstract of Twenty Cases of Intra-peritoneal**A. Rent closed*

No.	Reference.	Surgeon.	Age.	Cause.	Date after injury.	Condition of peritoneum.	Size and condition of rent in bladder.
1	Med. Rec. New York, 1887, xxxii, p. 781	Keyes	22	Run over by cart	22 hrs.	No signs of peritonitis; bloody fluid gushed out	Rent 1½ inches long
2	Lancet, June 28, 1888.	Halstrom	21	Not stated	21 hrs.	No details	Rent 1½ inches long
3	Arch. Gen. de Med., 1888, t. xxii, p. 22	A. Blum	28	Run over by waggon	40 hrs.	On opening peritoneum large quantity of fluid escaped; signs of peritonitis	Rent size franc piece
4	Med. Rec. New York, 1888, xxxiii, p. 632	Brown	—	Fell on corner of doorstep	22 hrs.	Blood-stained fluid found in peritoneal cavity; signs of peritonitis	Rent 1½ inches long; edges ragged
5	Pittsburg Med. Rev., 1888, ii, p. 55	Hitchcock	30	Blow	—	Extensive peritonitis	Rent 6 inches long
6	North American Practitioner, Chicago, 1889, i, p. 408	Herrick	28	Kicked by horse	23 hrs.	On opening peritoneum blood-stained fluid escaped	—
7	Indian Med. Gaz., Jan., 1889, p. 22	H. F. Stanton	Adult	Kicked in abdomen	50 hrs.	—	Extensive tear; posterior wall
8	Brit. Med. Journ., Jan. 26, 1889	R. Morrison	25	Distended cystitic; bladder gave way to slight injury	—	Signs of extensive peritonitis	½ inch rupture of pouch between ribs of bladder in upper posterior part
9	New York Med. Journ., Mar. 9, 1889	C. K. Briddon	Adult	—	3rd day	Signs of peritonitis	Rent posterior wall bladder

*Rupture of the Bladder published since 1888.*

*ry Sutures.*

Kind of sutures.	Treatment of peritoneum.	Incision in perineum.	Catheter in bladder.	After-treatment.	Remarks.	Result.
9 Lembert's sutures	Peritoneal cavity flushed	No	No	Catheter passed at intervals	Vomiting continued after operation; collapse set in	Died 18 hours after operation
9 Lembert's sutures	No details	—	—	No details	—	Recovered.
6 Lembert's sutures	Flushed with boiled water	No	Yes	Catheter taken out after 48 hours	After operation vomiting ceased, and peritonitis disappeared	Recovered.
Lembert's sutures	Irrigated with boiled water	No	Yes	Opium given	No leakage took place after suturing, when bladder was filled with Thiersch's solution	Died 16 hours after operation.
Lembert's sutures	Irrigated with boracic acid solution	No	No	—	—	Died
Lembert's sutures	Irrigated with boracic solution; glass drain used	No	No	Catheter passed at intervals	Peritonitis supervened	Died 49 hours after operation
Lembert's sutures	—	—	—	—	—	Died 24 hours after operation.
Sutures	Abdomen irrigated	—	—	—	—	Died 4 days after operation of general peritonitis
Lembert's sutures	Abdomen flushed out	No	No	—	—	Died 14 hours after operation.

No.	Reference.	Surgeon.	Age.	Cause.	Date after injury.	Condition of peritoneum.	Size and condition of rent in bladder.
10	New Zealand Med. Journ., Dunedin, 1888-90, viii, p. 158	Knight	49	Fell against corner of bench	54 hrs.	Fluid escaped on opening peritoneum; no peritonitis	Ragged rent 1½ inches long
11	Lancet, 1892, vol. ii, p. 197	Hulke	33	Butted in abdomen	72 hrs.	Several ounces bloody grumous fluid escaped on opening peritoneal cavity; extensive peritonitis	Rent 2½ inches long, posterior wall
12	Arch. für klin. Chir., Bd. xliii, Heft 1	Schlange	34	Run over by heavy waggon	24 hrs.	No peritonitis; blood-stained fluid found in cavity	Intra- and extra-peritoneal rent
13	Lancet, 1892, vol. i, Feb. 6	Page	—	Man fell across patient's abdomen	28 hrs.	Signs acute peritonitis	Stellate wound on back and upper part fundus
14	Lancet, 1893, vol. i, p. 413	Howard Marsh	34	Kicked in abdomen	80 hrs.	5 or 6 pints dark coloured urine mixed with blood escaped on opening cavity; signs of peritonitis	Ragged rent 2½ inches long behind and below fundus
15	Annals of Surgery, 1893, p. 653	Pilcher	23	Fell from second story of house	12 hrs.	Intra-peritoneal hæmorrhage; no peritonitis	Longitudinal rent about 1½ inches long
16	Lancet, 1894, vol. ii, p. 1032	Murphy	24	Run over by horse	2 hrs.	40 ounces fluid removed on opening abdomen; no peritonitis	Vertical rent extending from apex to trigone
17	The present case	Walsham	38	Man fell across patient's abdomen	25 hrs.	On opening peritoneum blood-stained fluid escaped, together with air that had been introduced in bladder; no signs of peritonitis	Vertical rent; edges slightly ragged; 2½ inches long; extending from apex to trigone

Kind of sutures.	Treatment of peritoneum.	Incision in perineum.	Catheter in bladder.	After-treatment.	Remarks.	Result.
Continuous suture	Peritoneal cavity thoroughly irrigated	No	Yes	Catheter removed on 4th day and passed at intervals	No peritonitis existed 54 hours after injury	Recovered.
Double row sutures	Peritoneal cavity flushed out	No	No	Urine drawn off at regular intervals	Vomiting returned after operation; peritonitis continued	Died 4 days after injury.
Lembert's sutures to intra-peritoneal rent	Peritoneum mopped out; not flushed; extra-peritoneal wound packed with iodoform gauze	No	No	Catheter passed at intervals	—	Recovered.
Lembert's sutures	Peritoneum flushed with solution boracic acid	No	No	—	—	Died before completion of operation.
20 fine silk Lembert's sutures	Abdominal cavity flushed	No	No	Catheter passed every 3 hours	Vomiting set in and patient sank gradually	Died 21 hours after operation of general peritonitis.
Lembert's sutures	Abdominal cavity sponged out and well irrigated	No	No	—	Hæmorrhage proceeded from torn superior vesical veins	Died 2 hours after operation from shock and anæmia.
15 catgut sutures to muscular coat only	Abdominal cavity washed out; glass drain tube used	No	Yes	Catheter removed at end of 8 days, and passed at intervals for one week	Diagnosis settled by injections of solution of boracic acid into bladder	Recovered.
14 fine silk Lembert's sutures	Abdominal cavity flushed with 1 in 8000 solution of Hyd. Perchlor., and finally with 2 per cent. solution boracic acid	No	No	Catheter passed every 4 hours for 24 hours, after which the patient passed urine of his own accord	Injection boracic acid solution gave negative results; diagnosis settled by inflating bladder with air, when liver dulness disappeared	Recovered.

*B. Rent not closed*

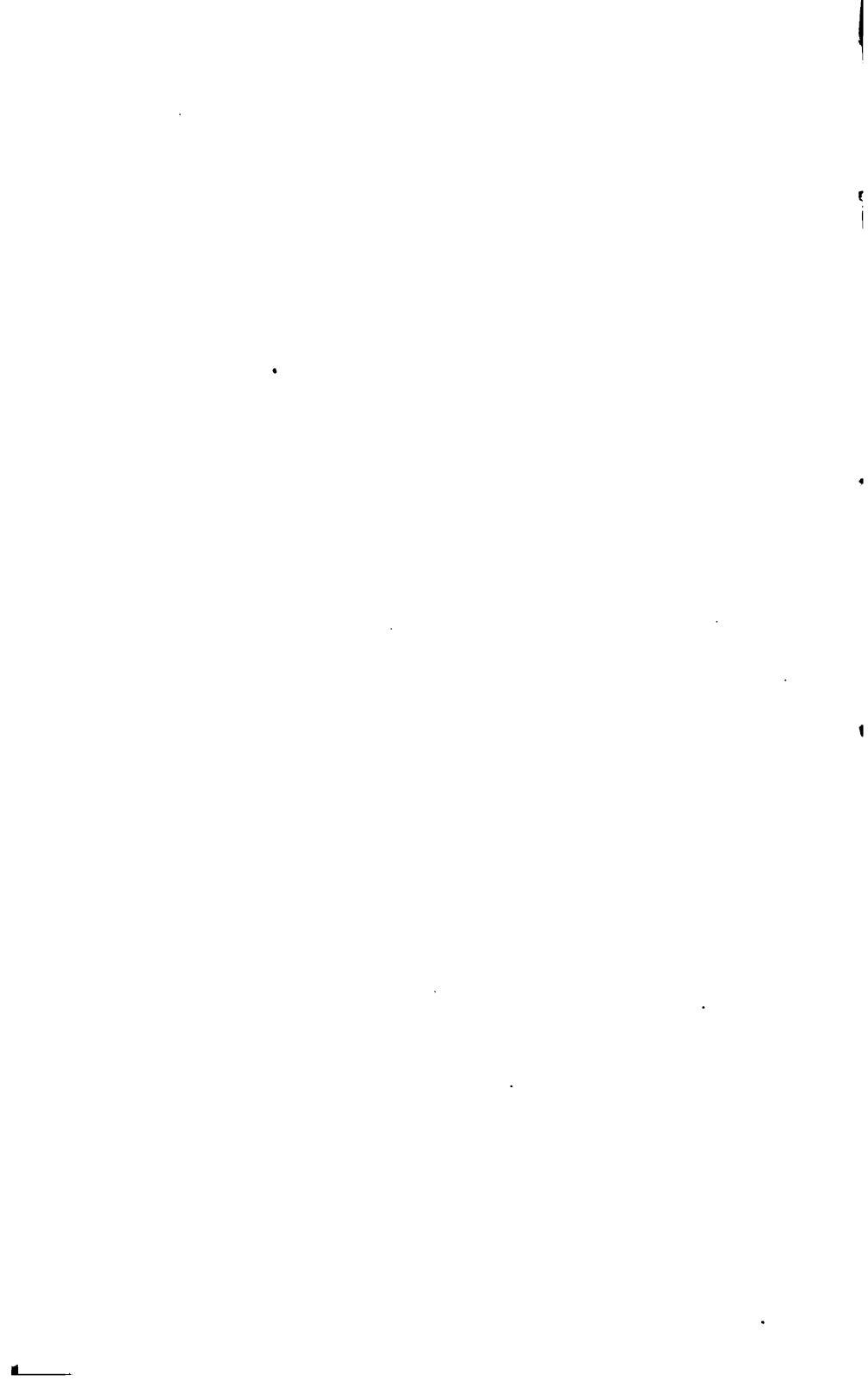
No.	Reference.	Surgeon.	Age.	Cause.	Date after injury.	Condition of peritoneum.	Size and condition of rent in bladder.
18	Lancet, Aug. 4, 1888, p. 208	W. H. Brown	20	Horse fell upon patient	11 hrs.	—	Rent posterior wall, far forward
19	Annals of Surgery, Dec., 1898	Kerr	33	Blow	8 hrs.	No peritonitis	Combined intra- and extra-peritoneal rupture
20	Deutsche Zeitschrift für Chirurgie, Bd. xxxi, Hefte 3 and 4	Rose	7	Blow	48 hrs.	No definite peritonitis; intestines slightly congested	Stellate rent on upper surface

(For report of the discussion on this paper, see 'Proceedings of the')

# ing Sutures.

Kind of sutures.	Treatment of peritoneum.	Incision in perineum.	Catheter in bladder.	After-treatment.	Remarks.	Result.
Wound inaccessible to suture	Abdomen irrigated; drain inserted	No	Yes	—	Wound could not be reached for applying sutures	Died 11 hours after operation.
Side of bladder sutured to pubes	Pelvis tamponed with iodoform gauze; bladder and Retzius space drained	No	No	Drain in bladder removed after firm adhesions had formed	—	Recovered.
Not sutured	Peritoneal cavity flushed; bladder drained through abdominal wound and packed with iodoform gauze	No	Yes	Abdominal drain removed as soon as adhesions had shut off abdominal cavity	—	Recovered.

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